

149

NIGERIA.

ANNUAL REPORT

ON THE

MEDICAL RESEARCH INSTITUTE

FOR THE YEAR

1926.



LAGOS:

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APPENDIX A.

ANNUAL REPORT OF THE MEDICAL RESEARCH
INSTITUTE, 1926.

BY

ANDREW CONNAL, M.D., D.P.H., D.T.M. AND H.

MEDICAL RESEARCH INSTITUTE,

YABA,

(NEAR LAGOS, WEST AFRICA),

21st March, 1927.

SIR,

I have the honour to present the Annual Report of the Medical Research Institute for 1926.

2. The following subjects received particular attention:—
Rat plague, yellow fever, prickly heat, tropical ulcer and markings on *Aedes argenteus*.

3. Plague in rats was found to be still in an acute form, no evidence of chronicity having been obtained.

There were further indications that yellow fever in West Africa is not identical with that disease in the Western Hemisphere.

The cause of prickly heat is considered to be a fungus belonging to the genus *Monilia*.

The main factor in the production of tropical ulcer is thought to be *Bacillus pyocyaneus*, of which organism a number of strains have been isolated.

The markings of *Aedes argenteus* have been proved to be very variable and no reasons for the creation of new varieties have arisen.

4. Other subjects dealt with are blackwater fever, malignant disease, black tongue, leprosy and mosquito-dissections.

5. Those who suffered from blackwater fever have invariably shown a malarial history.

Malignant disease has been shown to be by no means uncommon and the various forms of cancer to be not rare.

“ Black tongue ” was investigated and found to be due to a fungus of the oidium type, occurring in cases of relapsing fever.

Moogrol, in the treatment of both early and late cases of the three forms of leprosy, continued to be of great use.

Mosquito dissections showed that several local species were vectors of parasitic disease.

6. The Director was on leave from 12th February until 19th August as was also the Honorary Entomologist.

7. The Bacteriologist, Dr. E. C. Smith was on duty throughout the year, along with Mr. E. F. Hines, Laboratory Attendant.

8. Mr. F. W. Randall, appointed Laboratory Attendant, assumed duty on 4th February.

9. Mr. Hines did useful work in rat dissections, water analyses and media-making.

10. Mr. Randall was very helpful in section-preparation, in micro-photography and in the making of charts.

11. Dr. J. C. Paisley, on appointment to the West African Medical Service was seconded for rat plague work at Ereko, on his arrival in Lagos on 16th September. His assistance was of very great value.

I have the honour to be,

Sir,

Your obedient Servant,

A. CONNAL,

*Director of Medical Research
Institute.*

THE HONOURABLE

DIRECTOR OF MEDICAL AND SANITARY SERVICE,
LAGOS.

RAT PLAGUE IN LAGOS.

The dissection of the rodents caught by the official trappers or brought by volunteers was continued daily throughout the year.

Trapping was steadily pursued in the municipal area, but as concentrated efforts took place from time to time, in certain districts, and as the source of the rats brought by volunteers could not be determined, it would be misleading to group the findings, as has been done in previous reports, under the headings of Lagos, Iddo, Ebute Metta and Apapa. Consequently the results of the year's work are given under one heading, that of Lagos Municipal Area.

The original and unvaried scheme has been adhered to, that is, to dissect all rodents (with the exception of mice) after they have been identified and numbered, so that their species is known and their place of capture is traceable (except in the case of specimens from volunteers, who are purposely not asked for information). Smears were made from the spleen of each dissected animal, were fixed by heat and were stained with carbol thionin blue, after which each and all were examined microscopically, as has been the unbroken practice since the beginning of the outbreak of plague in Lagos, two and a half years ago.

The number of rodents and shrews thus examined in 1926 is 45,749, made up of 38,827 black rats, 4,003 brown rats and 2,919 shrews. One specimen of the "pouched" rat (*Cricetomys gambianus*), a "tree-bear" (*Hyrax* sp.) and two domestic cats were also dissected. During the latter part of the year it was decided to re-commence the examination of mice, with a view to checking the results obtained in the previous year when it appeared that plague in these animals was of infrequent occurrence.

The examination of the mice, which numbered 15,989, from the latter part of October until the end of December, showed that only four individuals suffered from plague, a negligible total at a time when from 1.70 to 6.16 *per cent.* of rats were infected.

The result of the mouse examination, therefore, is considered to have justified the course adopted, that of concentrating all investigation on the rats, thus saving much time and labour besides slides and stains. Including the mice, the full total of animals examined for plague is sixty-one thousand, seven hundred and thirty-two.

The monthly numbers and the percentage of infected animals are given in Tables I and II.

TABLE I.

	Black rat.	Positive.	Brown rat.	Positive.	Total rats.	Positive.
January ...	3,099	72=2·32%	317	4=1·26%	3,416	76=2·22%
February ...	2,516	55=2·18	302	4=1·32	2,818	59=2·09
March ...	2,908	44=1·51	257	2=0·77	3,165	46=1·45
April ...	2,800	15=0·53	233	nil.	3,033	15=0·49
May ...	3,525	19=0·53	270	nil.	3,795	19=0·50
June ...	3,422	52=1·51	247	4=1·61	3,669	56=1·52
July ...	3,897	65=1·66	386	3=0·77	4,383	68=1·55
August ...	3,627	71=1·95	450	9=2	4,077	80=1·96
September...	4,313	202=4·68	618	12=1·94	4,931	214=4·33
October ...	3,022	214=7·08	365	21=5·75	3,387	235=6·93
November	2,870	78=2·71	298	8=2·68	3,168	86=2·71
December ...	2,828	51=1·80	260	15=5·76	3,088	66=2·13
Totals ...	38,827	938=2·41	4,003	82=2·04	42,830	1,020=2·38

TABLE II.

	Shrews.	Positive.
January ...	133	% nil.
February ...	138	nil.
March ...	202	nil.
April ...	192	nil.
May...	192	nil.
June ...	189	nil.
July...	168	nil.
August ...	251	1=0·39
September ...	441	1=0·22
October ...	333	nil.
November ...	342	nil.
December ...	338	nil.
Totals ...	2,919	2=0·06

It will be seen from Table I that in 42,830 rats there were 1,020 infected with plague as compared with 15,989 mice of which four were infected. The period during which the mice were examined was 25th October, 1926, and 31st December, 1926, and in the same period 6,860 rats were dissected, of which number a total of 198 were shown to be plague-infected. It must not be thought that no mice were caught between January and October, for the daily bag of these was between two and three times larger than that of the rats.

The shrews (*Crocidura manni*) seldom exceeded twenty per day so that it was an easy matter to dissect them all. Table II gives the figures concerning them.

Chart I gives the percentage of infected rats and the total number of recorded cases of human plague for each week of the year. It shows the close relationship which thorough investigation alone can bring about. In other words, the chart proves that the system adopted for the detection of both human and rodent cases was efficient.

A minute examination of the rats was begun on 29th September, on a definite basis, after a month's survey of the usual conditions found on dissection, at this particular period, and compared with two years' previous experience. The dissection of one hundred rats per day was aimed at, although when time and material permitted, this figure was often exceeded. The total in ninety-four days was 9,178 a satisfactory figure considering that the number of rats brought in on Sundays seldom reached sixty.

Each rodent was fixed on a dissecting board by means of a fastener through each of the four limbs. After incision from chin to pubis, the skin was deflected to expose the cervical, axillary and groin groups of lymphatic glands. The abdomen and thorax were then opened and the state of the cavities and viscera noted.

Smears were taken from any and all enlarged, inflamed or suppurating lymphatic glands and cultures were made in all doubtful cases. Haemorrhages and pleural or peritoneal effusion were looked for. The condition of the liver and the spleen was observed and smears were taken from both organs. Smears were also taken from the lungs, the heart blood, the kidney, the suprarenal and the intestines when thought advisable.

As a result of this method of examination, it is possible to give certain facts and figures regarding rat plague in Lagos.

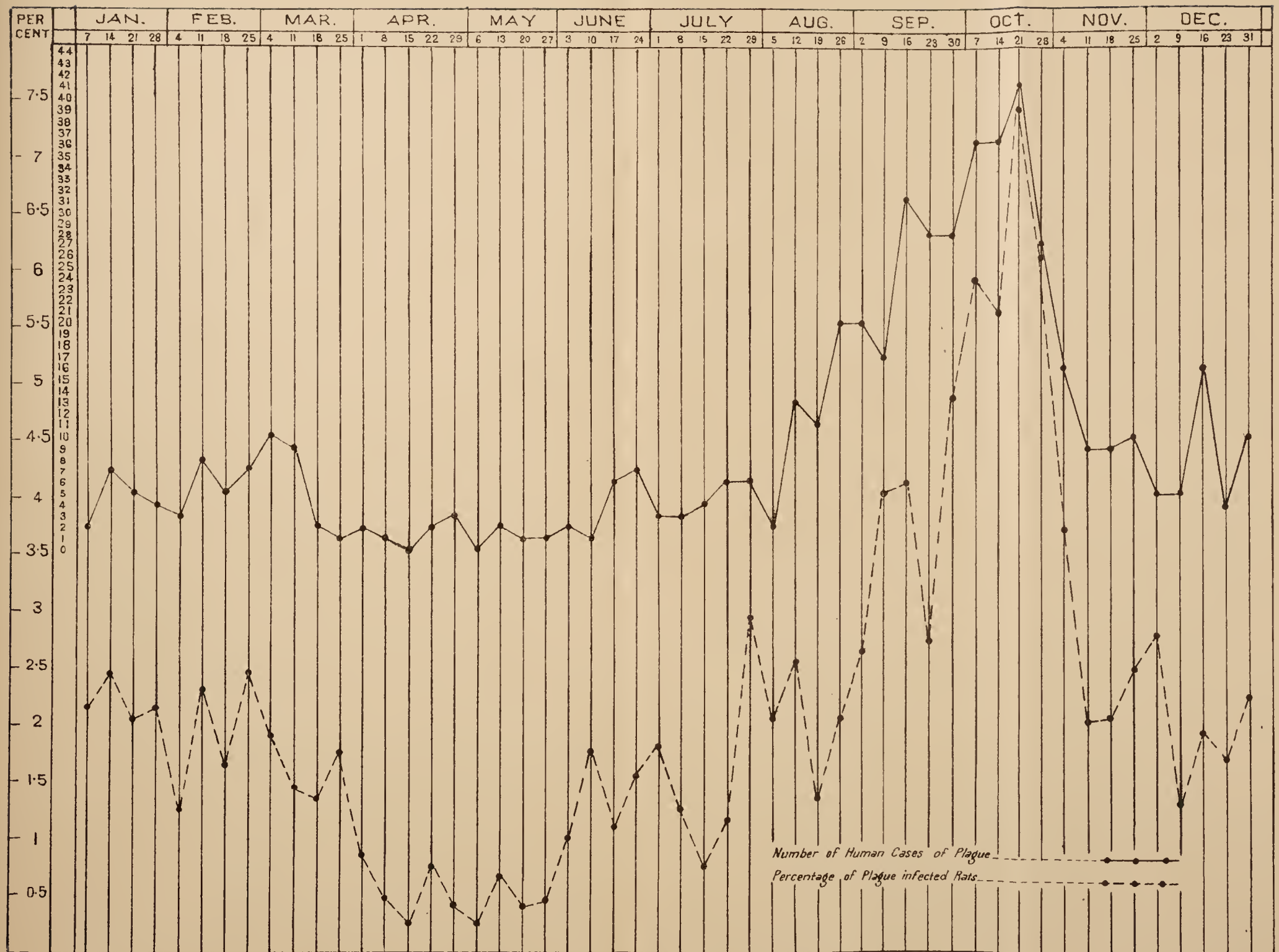
Dr. J. C. Paisley, who has done the bulk of the dissections, has drawn a very complete picture of the usual findings in *infected rats*, which is as follows:—

Rigor mortis.—This is generally well-marked in a fresh plague-rat, but it is also of common occurrence in apparently healthy rats.

Subcutaneous congestion.—It is an almost constant feature. It may be either local or general. Local congestion is always observed around the situation of the primary bubo, and it is common in all the glandular areas, *i.e.*, cervical, axillary and groin, even although no bubo is present. It is commonest in the neck, corresponding to the greater frequency of buboes in that region than elsewhere. General subcutaneous congestion is seen immediately on cutting the skin, as a rule, but if slight it may not be noticed until the skin is completely reflected off the thorax and abdomen. It has been observed in other conditions such as pregnancy, but its presence should arouse suspicion at the outset of the examination.

Subcutaneous haemorrhage.—This has not been found to be common. Haemorrhages have been frequently observed in the cervical region, but as the rats have been caught in break-back traps,

CHART I.



a traumatic cause is the more likely. Small points of haemorrhage may occur, though rarely, in the neighbourhood of the primary bubo. Haemorrhages in the flank have been observed in only two or three of the plague rats dissected.

Subcutaneous oedema either local or general, has not been encountered.

Enlarged lymphatic glands fall into two groups, namely primary buboes and secondary inflammations. The primary bubo, in its early stages is deeply congested and its colour is a dark reddish purple. There is considerable periadenitis, so that the gland is adherent to the surrounding tissue. When the bubo is cut across, haemorrhagic points are seen and the centre of the gland is softer than normal. At this stage the enlargement may be only slight. In the second stage, however, the increase in size is marked and the colour is yellowish white. The local congestion persists but is not so intense and the bubo is firmly fixed in the surrounding tissue. When cut across, the centre of the bubo is found to be semi-liquid and of a greyish or yellowish colour. The bubo is easily recognised at this stage, its size, colour and semi-liquid centre being distinctive. At both stages the bubo feels much harder to the forceps than an ordinary gland and it can often be detected by probing in the gland areas with this instrument.

Secondarily infected glands, that is, glands infected from the blood stream in septicaemic plague, are deeply congested, slightly enlarged and retain their normal consistence. They are not adherent to the adjacent tissues and local subcutaneous congestion is not so marked as in the case of a primary bubo. Microscopically they contain relatively few bacilli and these are typically bi-polar in their staining reaction, whereas the primary bubo is generally packed with bacilli which, especially in the second stage, include numerous involution forms. Buboes are most common in the cervical region and they are about equally as frequent in the axilla as in the groin. Buboes have not been found in the mediastinal or retro-peritoneal chain of glands.

Two or even more buboes may co-exist, such as one on each side of the neck, or one in the cervical and one in the axillary region.

The liver may show one of two characteristic changes. The commoner is manifested as a fine mottling. The other is speckled either finely or coarsely. Neither of these two conditions is, however, of frequent occurrence although when present they are practically pathognomonic. In many cases the organ shows only congestion and a considerable number exhibit no noteworthy change macroscopically.

In the case of "fine mottling," small stellate patches of congestion are scattered over the surface, alternating with greyish white areas, and with no distinct line of demarcation between them.

The "speckled" liver shows numerous well-defined greyish-white rounded patches of necrosis which vary in size. When fine, they are very numerous so that the organ has the appearance of having been dusted with pepper. They are, however, sometimes confined to the edges of the liver in which situation they are liable to be missed. In the coarser form of speckling, the necrotic spots are about the size of a pin head, in which case they are not so numerous as in the fine type.

The spleen is usually congested, swollen and protrudes further over the stomach than normally. But in adult rats, more particularly in the brown rat, the organ is frequently enlarged and congested, due to causes other than plague. In rare cases the spleen shows a speckled condition similar to that seen in the liver.

The kidneys and the suprarenals share in the general congestion but show nothing characteristic. For the purposes of naked eye diagnosis they are negligible.

The stomach.—Nothing abnormal has been noted in this organ.

The intestines.—In a certain proportion, not a large one, the intestines show extensive haemorrhage. The small intestine is the usual site, but in a few cases the large intestine is similarly affected. Further information on this interesting finding is given on a later page under its appropriate heading.

Peritoneal effusion.—An excess of blood-stained fluid in the peritoneal cavity has been frequently noted, but in every case trauma could not be excluded as a cause. An excess of clear peritoneal fluid has not been observed.

The lungs in plague rats are sometimes congested but broncho-pneumonic patches and pneumonic areas have not been recorded.

Pleural effusion is a very characteristic feature. The fluid is typically clear and straw-coloured and is usually abundant. Rarely, it is small in amount and very rarely blood-stained. An abundant clear pleural effusion has not been found in any non-plague rat.

Given a fresh plague rat (exceptionally they were brought fresh in Lagos) the characteristic signs are, marked rigor mortis, general subcutaneous congestion, an inflammatory condition in the cervical, axillary and groin areas, pleural effusion, enlarged spleen and a mottled or speckled liver. An analysis of the individual findings brings several interesting features to light.

The total of rats, black and brown, minutely examined is 9,178 of which there were 339 plague-infected.

Buboes, inflamed glands or gland abscesses were noted in 714 individuals. Plague bacilli were demonstrated in smears or by culture or by both means in 106, a proportion of roughly one in seven.

Positive cervical glands out-numbered all the others. The actual figures are:—

Cervical only	...	...	...	...	64
Axillary only	...	...	...	...	7
Groin only	...	...	...	...	7
All three sets	...	...	...	...	11
Cervical and axillary	...	...	...	...	9
Cervical and groin	...	...	...	...	7
Axillary and groin	...	...	...	...	1

The cervical glands, therefore, were affected in ninety-one cases, the axillary in twenty-eight and the groin glands in twenty-six.

There were bi-lateral cervical buboes in eleven cases and bi-lateral groin buboes in one.

In the total of 714 rats with enlarged glands, the gland contents were purulent in forty, *i.e.*, abscess-formation had occurred. The distribution was cervical twenty-nine, axillary six and groin five.

Of the twenty-nine cervical abscesses, eight were definitely due to plague. *Bacillus pestis* was present in the liver and spleen as well as in the pus in six, and was found only in the abscess in two.

As regards the six axillary abscesses, the plague bacillus was isolated in two, being noted in the liver and spleen as well as in the abscess in one, but only in the abscess in the other.

The five groin abscesses included two which were due to plague, the organism being shown in the liver and spleen as well as in the gland contents in both.

It will be noted that, in the total of forty gland abscesses there were twenty-eight (twenty-one cervical, four axillary and three groin) in which the purulent condition was due to some cause other than plague, inasmuch as neither smears nor cultures revealed the presence of *B. pestis*.

Of the remaining twelve abscesses, nine (six cervical, one axillary and two groin) showed the plague bacillus in the organs as well as in the pus, so that the condition in each case was one of septicaemic plague.

There were, therefore, only three cases (two cervical and one axillary) in which the organism was apparently confined to the abscess. It is not possible to state whether or not these were instances of chronic or "resolving" plague. The three individuals were brought by volunteers to the collecting station, whence the great majority of infected rats come. The explanation of the much greater number of plague infections in the rodents from the collecting stations than in those caught by the official trappers is thought to be due to the fact that the rats, being sick or dying, are readily caught and brought in for the monetary reward offered. It is possible, therefore, that the three rats under discussion were definitely sick and that had they lived a septicaemia would have supervened. In any case, up to the time of their death, the infection being confined to the gland was non-transmissible by means of fleas. Instances such as these appear to be negligible from the point of view of the spread of plague at a time when the septicaemic is the prevalent form of the disease in the rodents. Had they been found during a quiescent period their potential danger would have to be considered. Meanwhile the abundance of food available to rodents from the numerous street hawkers and booths renders it unlikely that surviving rodents are eating their own plague dead. It may be added that inflamed retro-peritoneal glands, indicative of an alimentary mode of infection, have not been encountered in any rat yet examined.

In addition to the positive findings in the glands recorded above (twelve cases in which abscess-formation had occurred) there remains another series of ninety-four cases in which plague bacilli were demonstrated in the glands. These cases were either early stages of the primary bubo or were secondarily infected from the blood stream. In seventy-nine instances the organism was present also in the spleen and liver. There were, therefore, fifteen cases, all primary buboes, in which the organism was present only in the gland. Here again it is not possible to state whether or not these last were cases of chronic or "resolving" plague, but for the reason that the bubo in each case was firm and surrounded by an area of congestion, it is probably correct to assume that the rat was killed at an early stage of an acute infection.

In the total of 339 plague infections, smears from the liver and spleen were positive in 321, while as already noted, the glands were positive in 106 cases. In thirty instances the liver and spleen were positive although the enlarged glands were negative. In eighteen cases, as recorded above, the glands were positive when no *B. pestis* could be found in the liver or spleen. In 201 cases there were no definitely enlarged glands, only the smears from the liver and spleen being positive. In a large number of this last group, however, the animal was more or less putrid. In two positive cases, decomposition was so far advanced that only a portion of the liver was available for examination.

The following are the actual figures:—

Glands, enlarged	714 cases.
Glands, positive	106 „
Glands, positive, organs negative ...	18 „
Glands, negative, organs positive ...	30 „
Organs, positive, glands not enlarged	201 „
Liver smears only	2 „

The liver.—Particular attention was devoted to the naked eye appearance of this organ, from 14th November onwards. During that period, 110 plague rats were dealt with. The laboratory notes made at the time of dissection and analysed later, afford the following information:—

Mottled liver	30 cases.
Yellow liver	14 „
Speckled liver	12 „
Normal appearance	6 „
Congested appearance	4 „
Not noteworthy	26 „
Putrid rats	18 „
Total	110 „

These figures show that in over fifty *per cent.* of the cases the appearances in the liver, typical of plague in rats, were lacking. It is striking that the “not noteworthy” livers rank next numerically to the mottled livers, the latter being generally a sure indication of the presence of plague.

Pleural effusion.—In the same total, namely 110 positives, pleural effusion was observed in thirty-five, was not observed in fifty-seven and was impossible to look for, because of advanced decomposition, in eighteen cases.

Dr. Paisley's general description of the post-mortem appearances in rat plague, and also the short analysis of the cases that follows, have been based on “The Diagnosis of Natural Rat Plague” (Reports on Plague Investigations in India, No. XI). The Journal of Hygiene, Vol. 7, No. 3, July 1907, pp. 324-358. The outstanding differences between the Indian findings in 1907 and the Lagos results in 1926 (dealing with the comparatively small number of 339 plague rats), are that buboes (and positive findings in their contents) are less common, that positive results from the spleen smears are more frequent, and that macroscopic changes in the liver are less noticeable in Lagos than they were in Bombay. Pleural effusion is practically equally important as an indication of infection. It would appear that the prevalent form of rat plague in Lagos is the septicaemic. Apparently either from the virulence of the organism or the virginity of the soil, the bacillus is able to penetrate the gland barriers without greatly disturbing these and so gains access to the blood-stream, in the majority of the cases. Again, there is no mention of extensive intestinal haemorrhage in the Bombay analysis. It should also be recorded that very few infections have been found in rats whose length, excluding the tail, is less than five inches. This is probably merely a question of harbourage for fleas.

Chronic plague.—This has already been discussed in connection with gland abscesses, and the conclusion arrived at is that there is no direct evidence that such a condition exists or has existed amongst the rats in Lagos. In the same number of the Journal of Hygiene, quoted above, Report No. XIX, “On the natural occurrence of chronic plague in rats,” pp. 457-471, the signs of chronic plague are divided into: A, the visceral type (1) splenic nodules and abscesses, (2) mesenteric abscesses, and B, the peripheral type (*i.e.*,

abscesses in the submaxillary, axillary, pelvic and inguinal regions). It should be noted that in the present report, the term "cervical" is used instead of "submaxillary" and "groin" is employed in place of "inguinal."

No cases of the visceral type have been observed in Lagos, nor have abscesses been encountered in the pelvic glands.

Between 5th September and 31st December, 1926, a number of wild rodents were kept in captivity for varying periods in order to see whether any which died or were killed had plague. Seventy-three black rats, eight brown rats and seven shrews were obtained alive. The first animal obtained, a black rat, died after eight days captivity, of septicaemic plague. In no other case were any signs of plague found. The periods were:—

Fifteen days, one rattus.
 Fourteen days, two rattus.
 Thirteen days, one rattus.
 Twelve days, one rattus.
 Eleven days, three rattus.
 Ten days, one rattus.
 Nine days, one rattus.
 Eight days, one rattus, three norvegicus.
 Seven days, four rattus, one norvegicus.
 Six days, two rattus, one norvegicus.
 Five days, one rattus.
 Four days, three rattus.
 Three days, fourteen rattus, one norvegicus.
 Two days, two rattus, one norvegicus, four shrews.
 One day, thirty-six rattus, one norvegicus, three shrews.

As regards the question of "resolved" plague, the following conditions were found in the spleen:—linear bands twenty-five, adhesions eighteen, necrotic areas ten and perisplenitis eight. In no instance were plague bacilli recovered from the lesions either in smears or by culture, but the safest test, that of guinea-pig inoculation, was not employed, for the reason that, in most cases, the signs were those of healing.

INTESTINAL HAEMORRHAGE IN PLAGUE RATS.

So far as can be ascertained, intestinal haemorrhage has not been recorded as occurring in plague rats. Small haemorrhagic spots or petechiae have been known to occur under the peritoneal coat or under or into the submucosa but the present remarks deal with massive bleeding into the lumen of the intestine.

The systematic routine examination of the rodents of Lagos, which was begun on 29th September, 1926, led to the finding of twenty-six cases of this condition, between that date and the end of the year. In every case in which it was noted, septicaemic plague was present. The macroscopic appearances are striking and are not readily overlooked. The entire or the greater length of the alimentary canal, with the exception of the stomach is distended and thin-walled so that the dark or bright red contents are distinctly seen. On opening the intestine, the contained matter is found to consist of blood-stained mucus mixed with a small amount of faecal matter and a number of worms. Ordinary heat-fixed smears of the contents generally showed a number of bi-polar stained bacilli and duplicate smears stained by Gram's method showed these bacilli to be Gram-negative. In most cases there was considerable admixture with other organisms such as make up the usual intestinal flora, but in a certain number *B. pestis* greatly predominated and in a few the smear showed what seemed to be practically a pure culture. Fresh smears and wet-mixed smears stained by Heidenhain's iron haema-

toxylin method showed no pathogenic protozoa. Cellular elements, *i.e.*, red corpuscles, pus cells and epithelial cells were comparatively few, so that the condition is not an ulcerative one. In no instance was the cause found to be traumatic, there being no free blood in the peritoneal cavity and no sign of injury to any of the abdominal organs. The possibility of an original alimentary source of the plague infection, of which the intestinal haemorrhage might have been the direct result was also considered, but the absence of retro-peritoneal buboes or inflamed mesenteric glands appeared to negative this supposition. It was therefore concluded that intestinal haemorrhage as met with in the rats suffering from septicaemic plague, is a late manifestation of that disease.

The occurrence of intestinal haemorrhage in twenty-six out of 339 plague rats, a percentage of 7.66, led to a consideration of its possible influence in the spread of human plague. In some epidemics, the cases are bubonic and septicaemic. In others, pneumonic cases occur as well, and in others again the cases are all pneumonic. The present considerations concern the second group, in which the pneumonic infection has been held to be due to a septicaemia following upon the bubonic form and its occurrence as a pure pneumonic form in other cases in the same epidemic, as due to direct contact by inhalation. It is presumed that the sick rat with intestinal haemorrhage, in Lagos, may deposit its blood-stained faeces on the earth floor of a native house, during the night. The usual sweeping which takes place on the following morning will distribute numerous plague bacilli in the air. The native habit of bending down whilst sweeping, using a short stiff home-made switch, renders the inhalation of some of the plague germs almost certain. It seemed that here was a probable explanation of some of the occasional cases of pneumonic plague which occur in the course of an epidemic in which most of the cases are of the bubonic type. With a view to ascertaining the infectivity of the blood-stained faeces of plague rats, the following experiments were undertaken :—

Experiment (1). 9.11.26—Intestinal contents of Rat No. 108,174 rubbed into scarified area, abdomen of each of two guinea-pigs. The infective material had been kept three days in the dark at room temperature, before use. Result. The animals each developed a small shotty gland in the inguinal region. Otherwise there was no sign of infection and both animals remained healthy. This result was considered to be negative.

Experiment (2). 10.11.26—Similar to Experiment (1), using material from Rat No. 108,508, but in this case the intestinal contents had been kept for only one day before use. Result as in Experiment (1).

Experiment (3). 18.11.26—Material from Rat No. 108,710, mixed with sterile dust on day of capture, 11/11/26, kept in dark at room temperature for one week. Mixture then suspended in sterile saline and injected into the trachea (after cutting down and exposing it) of Monkey No. 1. (*Cercoptes tantalus*). The material was also rubbed on two guinea-pigs as in Experiments (1) and (2). Results. Negative.

Experiment (4). 19.11.26—Faeces of Rat No. 109,797, mixed with sterile dust at once, allowed to dry for two hours in the dark, at room temperature, then suspended in sterile distilled water and injected (as in Experiment (3)) into exposed trachea of Monkey No. 2. (*C. tantalus*). One guinea-pig also inoculated, as before. Two c.c. of the suspension were used for the monkey.

The monkey died in seventy-two hours. P.M. 22.11.26—General subcutaneous congestion on section. Cervical, axillary and groin glands swollen and congested. Left lung showed a fibrinous pleural exudate and both lobes showed extensive areas of red hepatisation. Right lung, middle lobe also showed red hepatisation. Both liver and spleen enlarged, congested and firm. Smears from the glands, the lungs, the liver and the spleen showed *B. pestis*, these being particularly numerous in the lung smears. The guinea-pig behaved as in Experiment (1).

Experiment (5). 24.11.26—Bloody faeces of Rat No. 110,279, kept in dark at room temperature, twenty-four hours, smeared on scarified area, abdomen, two guinea-pigs. Result as in Experiment (1).

Experiment (6). 1.12.26—Intestinal contents of Rat No. 110,945, mixed with sterile dust on day of capture (30.11.26) suspended in sterile saline after twenty-four hours in the dark at room temperature and injected into the exposed trachea of Monkey No. 1 (same animal as was used in Experiment (3)). Two c.c. of the suspension were used. The animal died in ninety-six hours. P.M. 5.12.26—General subcutaneous congestion on section, general glandular congestion. Excess of free fluid in both pleural cavities with some flakes of lymph in the left. Left lung, lower lobe shows extensive patchy consolidation, greyish-red in colour. Right lung in the posterior portion of the upper and lower lobes shows a similar appearance. Liver and spleen congested. Smears from both lungs showed innumerable *B. pestis*, but the organisms were few in the liver and spleen.

Experiment (7). 4.12.26—Material from Rat No. 111,313, mixed with sterile dust and saline after twenty-four hours in the dark at room temperature. Injected through the exposed trachea of Monkey (3) (*C. patas*). Three c.c. of the suspension were used. The animal died in forty-eight hours.

P.M. 6.12.26—General subcutaneous congestion but no definite gland involvement. Both lungs intensely congested but no consolidation. Sub-pleural haemorrhages, right apex. Slight excess pleural fluid. Spleen and liver markedly congested. Smears from the lungs, heart blood, liver and kidney showed very few organisms, only an odd *B. pestis* being seen. The spleen smear, however, showed a few more.

Experiment (8). 8.12.26—Intestinal contents, Rat No. 111,408, kept twenty-four hours then allowed to dry in sterile dust in the dark at room temperature for three days, then sprayed as dry dust over nostrils and open mouth of Monkey No. 4 (*C. mona*). Result negative.

Experiment (9). 9.12.26—Material from Rat No. 111,856, dried in sterile dust for twenty-four hours in the dark at room temperature and sprayed as in Experiment (8) on Monkey No. 5 (*C. patas*). Result negative.

Experiment (10). 20.12.26—Intestinal contents, Rat No. 113,324, allowed to dry in sterile dust for three hours in the dark at room temperature. Sprayed as dry dust over mouth and nostrils of Monkey No. 5 (same animal as in Experiment (9)) and sprayed as liquid in sterile water over mouth and nostrils of Monkey No. 4 (same animal as in Experiment (8)). Result in both cases negative.

Experiment (11). 21.12.26—Intestinal contents, Rat No. 113,411, kept twenty-four hours in the dark at room temperature, smeared on scarified area, abdomen, each of two guinea-pigs. Result as in Experiment (1).

These experiments indicate that pneumonic plague can be transmitted to monkeys if the intestinal contents mixed with sterile dust and water are not kept longer than twenty-four hours and if they are injected directly into the trachea. Ordinary spraying methods either as dry dust or as a liquid suspension failed to infect monkeys on the four occasions on which the attempt was made. It should be stated here, however, that at a later date (8.1.27) the disease was successfully transmitted to Monkey No. 6 by liquid spraying. The failure to infect guinea-pigs through a scarified area on the skin of the abdomen and the several failures with monkeys as recorded above are not readily explained. But as it has already been noted, the intestinal contents in some cases show a preponderance of organisms other than *B. pestis* and the infectivity of the material may depend partly on the original number of *B. pestis* present, partly on the length of time after the death of the rat (or the deposition of the faeces) and partly on the action of the other bacilli on the plague organism.

The experiments are being continued and attempts are being made to infect healthy rats by feeding them on bread soaked in infected faeces. The results will be communicated in collaboration with Dr. Paisley and Corporal Bowrey, R.A.M.C.

Meanwhile, it is considered that there is a real possibility of human pneumonic plague being contracted by means of infective rat faeces.

EXAMINATION OF WILD RODENTS IN THE MUNICIPAL AREA.

In connection with the examination of wild rodents, Professor A. S. Pearse of the West African Yellow Fever Commission (International Health Board) has supplied the following information:—

“ In connection with my work on Nigerian rodents, I have examined 375 specimens of seventeen species collected from Yaba north to the Lagoon. In all of these I have looked for evidence of plague such as injected glands, enlarged spleen, etc., and in the few cases that looked suspicious I have made smears that were stained with carbol-thionin. In none of these cases did I find plague.”

THE ECTOPARASITES OF THE LAGOS RODENTS.

From 29th September until the end of the year, all the dead rats brought for examination were first of all well shaken, individually, in the disinfectant fluid in which they arrived. The liquid was then poured through a fine-meshed sieve and the retained matter was afterwards washed under a tap into a white basin from which the ectoparasites were readily picked out. A certain number of fleas must have been missed by this method, but the main consideration is that dead rats were being dealt with. From the point of view of obtaining reliable data on the number of fleas per rat, the findings are unsatisfactory. For it is known that rat fleas desert their host as soon as there is realisation that it is no longer possible to obtain a blood meal. Only a very small proportion of the rats received were recently dead and a considerable number were more or less putrid, and therefore in all probability barren of fleas.

In the light of the foregoing explanation, the following figures are given.

The total number of ectoparasites collected in three months from dead rats (9,178) was 1,294, made up of 1,129 fleas, 159 *Laelaps*, five ticks and one *Anopleura*.

The fleas therefore averaged over the whole period in the neighbourhood of one flea to eight rats.

From 29th September to 31st October, 3,036 rodents were dealt with, the term “ rodents ” for the purposes of this account referring exclusively to black and brown rats. From these, 644 fleas were recovered, an average of about one flea to four rats.

In November there were 3,126 rats and 329 fleas, the proportion being nearly one flea to ten rats.

In December, 3,016 rats were examined and 156 fleas were obtained, giving a ratio of one flea to nineteen rats.

Only two species of fleas were obtained, *Xenopsylla cheopis* and *Xenopsylla brasiliensis*. In the first period there were 432 *X. cheopis* and 212 *X. brasiliensis*, a proportion of almost exactly two to one.

In November, there were 217 *X. cheopis* and 112 *X. brasiliensis*, again closely approximating to a proportion of two to one.

In the third period (December) there were 111 *X. cheopis* and 45 *X. brasiliensis*, so that the relation was distinctly more than two to one. It will be observed that although the figures admittedly do not represent the natural number of fleas per rat, they fall into line with accepted beliefs in two ways. In the first instance fleas were most numerous per rat in October, when the incidence of rat plague was highest, and were least numerous per rat in December when the

incidence of rat plague was the lowest of the three months under review. Further, they show a definite preponderance of the known plague-vector, *X. cheopis* over the other flea *X. brasiliensis*.

With regard to live rats, seventy-seven black and eight brown were received at the Research Institute. The custom invariably followed was to put each cage at once into a muslin bag, to prevent the escape of fleas. The bag was opened once a day to allow of feeding the rats. After the expiry of ten to fourteen days (in many instances the rats survived a less period) chloroform was poured over the muslin bag. When a sufficient length of time had elapsed to ensure the death of the rodents and the parasites, a flea count was made. The inside of the muslin bag as well as the fur of the rat was carefully searched. The following figures therefore may be taken to represent actual conditions.

In September, fourteen black rats were received alive. They yielded twenty-seven fleas, *X. cheopis* fourteen and *X. brasiliensis* thirteen, an average of two fleas per rat. In October, three black and two brown rats were obtained. Not a single flea was found.

In November, only one live rat came to hand and no fleas were found upon it.

In December, a steady supply of rats came in. There were fifty-nine black and six brown rats. From these, twenty-seven fleas were collected, *X. cheopis* eighteen and *X. brasiliensis* nine, a ratio of one flea to two rats.

Table III gives the figures concerning the fleas. It includes the collections from both dead and live rats.

TABLE III.

				September.	October.	November.	December.	Total.
<i>X. cheopis</i>	♂	...	...	16	266	136	72	490
				12	152	81	57	302
<i>X. brasiliensis</i>	♂	...	...	15	136	71	34	256
				11	63	41	20	135
Total	...	...	...	54	617	329	183	1,183

Males	...	...	746	<i>X. cheopis</i>	...	...	792
Females	...	...	437	<i>X. brasiliensis</i>	...	...	391

The highest collections of fleas from dead rats were made on two days in October, namely fifty-six fleas from 100 rats and twenty-five fleas from thirty-nine rats. At the other end of the scale, no fleas were obtained from 123 rats on a day in December.

Of the live rats, on one occasion five fleas and on another, four fleas were taken from a single rat.

All specimens of *X. brasiliensis* were dissected and examined. In no instance, however, was the number of plague bacilli sufficient to occlude the alimentary canal at the entrance to the stomach, although in a few cases plague bacilli were found in stained smears from that region. With regard to the other ectoparasites, there were 191 specimens of *Laelaps echidninus*.

The ticks, and the *Anopleura* await identification. The Honorary Entomologist was responsible for the identification of the ectoparasites.

EXAMINATION OF SPLEEN SMEARS FROM PLACES OUTSIDE LAGOS.

Smears were received from Abeokuta, Ibadan, Ijebu-Ode and Kano. The total number was 2,405 smears.

Abeokuta sent the largest number, namely 1,304, all of which were negative. There were 878 rat spleen smears from Ibadan, none of which showed *B. pestis*. From Ijebu-Ode and district 212 smears were examined, in ten of which the presence of *B. pestis* was demonstrated.

Eleven smears were received from Kano, all of which were negative.

BLACKWATER FEVER.

More or less complete reports have been received, concerning twenty cases of blackwater fever which occurred in Nigeria during the year.

All the cases occurred in male adults, none of whom hailed from Africa. The race is given as—

Australian in three cases.
English in ten cases.
Irish in two cases.
Scottish in five cases.

Officials and non-officials were equally divided, numbering ten each. The Railway supplied four cases, two locomotive drivers, an engineer and a workshop artisan. The other officials were a Superintendent of Education, a diver attached to the Harbour Works, a Captain in the West African Frontier Force, an Assistant in the Secretariat, a Collector of Customs and a Senior Medical Officer. The unofficial group included five employed in general trade, three mining engineers, a timber worker and a Roman Catholic Priest.

Thirteen cases occurred in the Southern Provinces, namely six in Lagos, two in Sapele, and one each in Abakaliki, Asaba, Ijebu-Ode, Onitsha and Port Harcourt.

There were seven cases in the Northern Provinces, two at Jos, and one each at Jemaa, Kano, Makurdi, Minna and Offa.

As regards the time of year, there were three cases in January, one in February, three in March, one in April, two in May, two in June, one in July, one in August, two in September, one in October, one in November and two in December.

The age of the patients is noted as 25, 26, 27 (2), 32, 36, 38, 39, 42 (2), 44 (3), 45, 46, 47, 48, 50, 51 and 55 years.

The total West African service (which in most cases includes leave) was 25, 22, 20 (2), 16, 15, 13, 12, 8, 6 (2), 5, 4 (2), 3, $2\frac{1}{2}$, 2, $1\frac{1}{4}$ (2) and "some" years.

Other tropical experience was possessed by Case (3) "much in tropics" (three years in West Africa), Case (5) Costa Rica three years (four years in West Africa), Case (14) Ceylon three years (West Africa two years), Case (15) South and East Africa and Chili ? years (six years in West Africa) and Case (20) India ? years (sixteen years in West Africa).

The length of the present tour of service was 5, 4, 3, $2\frac{1}{2}$, $1\frac{1}{2}$ (2), $1\frac{1}{4}$ (2), 1 (2), $\frac{3}{4}$ (2), $\frac{1}{2}$ (4), $\frac{1}{4}$ (2), and $\frac{1}{8}$ (2) years.

Quinine was taken prophylactically, with regularity (presumably daily) in three cases, on alternate days in one case, three times a week in one case, was admittedly occasionally missed from the

daily routine in only two cases, was seldom taken in two cases, was never taken in two cases and was taken at irregular intervals in ten cases.

A history of previous malarial attacks was obtained in every case. These attacks are described as frequent in one, repeated in three, many in four and several in twelve.

The facts relating to quinine and the onset of blackwater are :—

Case (1).	Hydrochloride,	grains 5,	5 hours before onset.			
.. (2).	..	.. 20,	12	..	..	..
.. (3).	..	.. 10,	8	..	..	..
.. (4).	..	.. 10,	few	..	..	..
.. (5).	..	.. 10,	7	..	..	..
.. (6).	Sulphate,	.. 1 $\frac{1}{4}$,	few	..	..	..
.. (7).	Hydrochloride,	.. 5,	16	..	..	..
.. (8).	?	.. 10,	72	..	..	..
.. (9).	?	.. 10,	7	..	..	..
.. (10).		NONE				
.. (11).	?	grains 30,	few	..	..	..
.. (12).	?	.. 5,	22	..	..	..
.. (13).	?	.. 5,	17	..	..	..
.. (14).	?	.. 10,	11	..	..	..
.. (15).	?	.. 5,	5	..	..	..
.. (16).	?	.. 5,	23	..	..	..
.. (17).	Hydrochloride,	.. 5,	1 hour	..	..	..
.. (18).	..	.. 5,	12 hours	..	..	..
.. (19).	?	.. 5,	few	..	..	..
.. (20).	?	.. 6,	12	..	..	..

Case 1.—Five grains quinine hydrochloride was the only dose taken.

Case 2.—Took four five-grain tabloids of quinine hydrochloride during the night, blackwater appearing about twelve hours after the last five-grain dose.

Case 3.—Took ten grains of the hydrochloride in one dose.

Case 4.—Had been taking ten grains hydrochloride daily in five-grain doses for a few days.

In all the above cases, the Medical Officer was called in after the appearance of blackwater.

Case 5.—Subtertian malaria diagnosed 5th March, 1926, given ten grains quinine hydrochloride in solution. 6th March, 1926, given fifteen grains in solution. 7th March, 1926, given ten grains in solution in morning and again in evening. 8th March, 1926, given ten grains in solution in the morning. Blackwater appeared 7 $\frac{1}{2}$ hours later.

Case 6.—On 7th March, 1926 had “ fever ” and took two pills of Esanofele. Fever continued and on 8th March, 1926, took another two pills of Esanofele. On 9th March, 1926, consulted Medical Officer who prescribed mixture which contained quinine sulphate in 1 $\frac{1}{4}$ grain doses to be taken hourly. Six doses were taken but as there was persistent vomiting, most of it probably was rejected.

Case 7.—Took five grains quinine hydrochloride on day before onset.

Case 8.—Took ten grains quinine in tabloid form, three days before the appearance of blackwater.

Case 9.—Took ten grains quinine in tabloid form seven hours before onset.

Case 10.—Took no quinine.

Case 11.—For four days before onset took thirty grains quinine daily.

Case 12.—Took his usual daily five-grain tabloid of quinine.

Case 13.—Had taken five grains quinine daily for “about a week.”

Case 14.—Admitted to hospital 26th August, 1926, and given ten grains quinine in liquid form. On 27th August, 1926, he was given ten grains in liquid form three times, the last dose at 6 p.m. Blackwater, eleven hours later.

Case 15.—Subtertian malaria diagnosed 8th September, 1926, given five grains quinine hydrochloride in solution in the morning and again in the evening. On 9th September, 1926, the same amount was given in the same dosage, last dose at 6 p.m. Blackwater appeared five hours later.

Case 16.—Took a five-grain tabloid of quinine.

Case 17.—Subtertian malaria diagnosed 29th October, 1926, and given five grains quinine hydrochloride in liquid form twice. On 30th October, 1926, this dose was given three times, the last being taken one hour before blackwater appeared.

Case 18.—On 27th October, 1926 took ten grains quinine hydrochloride in tabloid form daily for a week, then five grains daily until 10th November, 1926, when he restarted ten grains daily. Last dose midday 16th November, 1926. Blackwater early morning 17th November, 1926.

Case 19.—Took the usual five-grain tabloid.

Case 20.—Took six grains in powder form $11\frac{1}{2}$ hours before onset.

MODE OF ONSET, SYMPTOMS AND COURSE.

Case 1.—A.R. Felt ill 4th January, 1926, rigor in evening and blackwater seen at midnight. Severe headache, backache, vomiting, jaundice, enlarged spleen. Urine did not clear. Temperature between 100° and 104° F. for three days, then normal. Suppression set in on fourth day and death on sixth day.

Case 2.—K.S. Shivering, backache, malaise, evening of 9th January, 1926. Blackwater 2 p.m. 10th January, 1926. Jaundice, vomiting, enlarged spleen. Urine did not clear. Suppression set in on fourth day, death on tenth day.

Case 3.—A.C. Felt ill, 11th January, 1926. Blackwater 12th January, 1926. Vomiting, jaundice, backache. Urine cleared in three days. Not seen by Medical Officer until 15th January, 1926 when temperature, pulse and urine normal.

Case 4.—J.R. Rigor on 11th February, 1926 (unwell for previous ten days) blackwater on 12th February, 1926. Epigastric pain, deep jaundice. Bilious vomiting. Repeated haematemesis on 13th February, 1926. Stools loose and melaena on 14th February, 1926. Liver and spleen enlarged. Suppression set in on third day, death on fifth day.

Case 5.—J.M. Fever with occasional vomiting for fourteen days. Blackwater on 8th February, 1926, rigor on 9th February, 1926. Vomiting and jaundice slight.

Febrile temperature subsided in seven days, urine cleared in six days.

Case 6.—T.R. On 7th March, 1926 had fever, headache and vomiting. Took two pills Esanofele. On 8th March, 1926 symptoms continued, took two other pills same drug. On 9th March, 1926 consulted Medical Officer, who prescribed quinine sulphate hourly in doses of $1\frac{1}{4}$ grains. Blackwater appeared that evening. Admitted to hospital two hours later. Urine thereafter was clear. There was slight jaundice but no vomiting. The spleen was enlarged.

Case 7.—B.E. Felt seedy on 22nd March, 1926. Blackwater on 23rd March, 1926. Backache, vomiting, diarrhoea and jaundice. Carried one day's journey to hospital. Suppression had then set in. Died 25th March, 1926.

Case 8.—W.P. Felt off colour, 25th April, 1926. Felt better next day but vomited at night. Remained in bed 27th April, 1926 but got up in evening. During the night had rigors and vomiting, temperature 103° F. Seen by Medical Officer 28th April, 1926 just after blackwater had appeared. Jaundice, headache, nausea, tarry stools. Urine cleared on 1st May, 1926 and temperature became normal on 30th April, 1926.

Case 9.—H.F. Felt unwell on 4th May, 1926 vomited in evening and early next morning noticed blackwater. Urine nearly clear by evening, but darker again on 6th May, 1926. Cleared entirely on 7th May, 1926.

Case 10.—P.C. Had fever intermittently for five months. Did not take quinine. On 3rd May, 1926 had fever and shivering in evening. On 5th May, 1926 there was still vomiting and fever and blackwater was noticed in the evening. He was first seen on 7th May, 1926 and admitted to hospital. There were then epigastric pain, vomiting and some jaundice, but the temperature was normal. Haemoglobinuria disappeared on the ninth day of illness but vomiting persisted. Albuminuria which was at first heavy, remained as a haze, during which time there was considerable vomiting, sometimes stercoraceous, and ascites developed. The patient gradually grew weaker and on the eighteenth day began to show a rise of temperature up to 100° F. in the evenings. On the twenty-second day the temperature rose to 103° F. and he died in the evening.

Case 11.—E.A. Headache, vomiting and fever from 25th June, 1926 until 29th June, 1926. No rigor, blackwater evening of 29th June, 1926. Spleen enlarged, sclerae faintly yellow, no febrile disturbance. Urine cleared in thirty-six hours and size of spleen rapidly diminished under quinine administration.

Case 12.—A.F. Felt unfit, evening of 14th June, 1926 with backache, no actual rigor but slight chill. Blackwater seen on morning of 15th June, 1926. Slight nausea, spleen enlarged and tender, liver tender, some jaundice. There was never any vomiting. Temperature fell from 100.8° F. on 15th June, 1926 to normal on 16th June, 1926 and urine cleared on fourth day.

Case 13.—Feeling out of sorts for a week during which the urine gradually darkened in colour. When Medical Officer was consulted (30/7/26), it was dark red. On the following day it was clear. Quinine was exhibited on 4th August, 1926 and gradually increased in dosage until 10th August, 1926 when there was a recurrence of haemoglobinuria lasting twelve hours. The temperature on 30th July, 1926 was 101° F. and became normal next day. On 10th August, 1926 it was again 101° F. becoming normal on 11th August, 1926. There was never any vomiting or any discomfort, but there was marked jaundice. Patient had a double aortic murmur.

Case 14.—A.B. Complained of being seedy for some time. Admitted to hospital, evening of 26th August, 1926 and under quinine treatment the temperature became normal. On the early morning of 28th August, 1926 blackwater appeared and the temperature rose to 102.6° F. Slight jaundice, liver and spleen enlarged. Headache, some vomiting. Temperature normal on 29th August, 1926, no further vomiting, no discomfort. Urine clear on 30th August, 1926.

Case 15.—A.M. Admitted to hospital with subtertian malaria on 8th September, 1926. The temperature was then 103.4° F. On 9th September, 1926 the temperature was normal but blackwater appeared in the evening. On 10th September, 1926 the temperature was 102.4° F. Spleen tender and palpable. No vomiting, slight jaundice. The urine cleared during the morning but was red again in the afternoon, clearing finally in the evening.

Case 16.—J.F. Had a malarial attack, 11th September, 1926 to 14th September, 1926. Black urine noticed in evening of 16th September, 1926. No rigor, some nausea and pains in loins. Spleen enlarged and vomiting slight. Urine cleared on third day.

Case 17.—D.D. Malarial attack on 29th October, 1926, blackwater evening of 30th October, 1926. Backache and headache, no rigor. Vomited once. No jaundice. Temperature 103° F. on 29th October, 1926, 102° F. on 30th October, 1926, normal next day. Urine cleared in eight hours.

Case 18.—E.W. Headache, nausea and pyrexia on evening of 16th November, 1926, which continued until early hours of 18th November, 1926, when blackwater appeared. Temperature 101.2° F., no rigor, spleen enlarged, vomiting, jaundice. Urine cleared after three days, vomiting ceased after second day, but intermittent temperature persisted six days.

Case 19.—F.M. Lassitude for four days, then rigor and blackwater on night of 18th December, 1926. Jaundice observed next day, journeyed to hospital arriving there on 20th December, 1926, temperature sub-normal and urine black. Epigastric and lumbar pain, headache and frequent vomiting. Urine did not clear. Suppression set in and temperature rose to 102.6° F. on 22nd December, 1926. Patient became unconscious and died on 23rd December, 1926.

Case 20.—S.G. Seedy for four days. Had a rigor on night of 28th December, 1926 and blackwater appeared in early morning of 30th December, 1926. Had loin pains and nausea. Both liver and spleen enlarged and tender. Temperature 100.4° F. Temperature normal next day and urine cleared.

There was a fatal result in six cases, the cause of death being suppression of urine in five. In the sixth case there was evidence of abdominal disease, the indefinite nature of the lesion and the precarious state of the patient preventing operative interference.

The duration of haemoglobinuria in the cases which recovered was eight hours, nine hours, twelve hours, one day, two days (4), three days (4), four days and six days. A remission took place in two cases and a relapse in one case. Blood smears were examined by the Medical Officer except in one case. The results are tabulated below :—

CASE.	DAY OF EXAMINATION.	RESULT.
(1)	?	No parasites seen.
(2)	no examination.	
(3)	?	No parasites seen.
(4)	second day	No parasites seen.
(5)	Three days previous to onset of blackwater.	Subtertian rings.
(6)	first day	Subtertian rings.
(7)	?	No parasites seen.
(8)	Two days after urine cleared.	Subtertian rings.
(9)	second day	No parasites seen.
(10)	fifth day	No parasites seen.
(11)	first day	No parasites seen.
(12)	first day	No parasites seen.
(13)	second day	No parasites seen.
(14)	first day	No parasites seen.
(15)	Day previous to onset of blackwater.	Subtertian rings.
(16)	second day	No parasites seen.
(17)	Day before onset of blackwater.	Subtertian rings.
(18)	first day	No parasites seen.
(19)	third day	No parasites seen.
(20)	first day	No parasites seen.

Blood smears were kindly sent to the Medical Research Institute from six cases. Neither parasites nor pigment were seen in any.

A differential leucocyte count was made in each case, and the Arneth formula was also obtained, the count being based on the enumeration of 500 white cells and the formula on the examination of 500 polymorphs. The findings are given in tabular form.

TABLE IV.
DIFFERENTIAL LEUCOCYTE COUNTS.

Cases.	Day.	P.	S.	L.	M.	E.	T.	Ma.	My.	Ery.	Vac.	N.R.
7.	second	60·8	18·	2·8	17·2	—	0·6	0·2	0·4	2·	9·	1·
10	fifth	73·6	11·2	2·2	4·4	4·4	3·4	0·4	0·4	—	—	—
16	first	60·	20·8	2·	12·	2·	3·2	—	—	—	—	—
	second	64·8	14·4	2·8	14·4	0·8	0·8	—	2·	—	—	7·
17	second	51·2	19·6	2·8	24·8	—	0·8	—	0·8	—	—	—
	third	34·	41·6	3·2	14·	4·8	2·4	—	—	—	—	—
19	second	84·4	6·8	1·6	6·2	0·2	0·6	—	0·2	1·	3·	—
	third	82·4	9·	0·8	5·6	—	2·	—	0·2	—	1·	3·
	fourth	77·8	7·8	1·8	7·4	0·6	2·6	—	2·	—	—	—
20	second	53·6	12·6	2·	24·6	5·2	1·4	—	0·6	—	3·	2·

In the above Table, P = polymorphs, S = small lymphocytes, L = large ditto, M = mononuclears, E = eosinophils, T = transitionals, Ma = Mast cells, My = myelocytes, Ery = erythrophages, Vac = vacuolated mononuclears, N.R. = nucleated red cells.

TABLE V.
ARNETH FORMULÆ.

Cases.	Day.	I.	II.	III.	IV.	V.
7	second	65·6	26·8	6·8	0·8	—
10	fifth	46·8	34·4	13·2	5·6	—
16	first	56·2	32·4	10·2	1·2	—
	second	50·4	28·2	18·	3·4	—
17	second	86·2	12·4	1·4	—	—
	third	84·4	11·2	4·4	—	—
19	second	71·6	22·8	5·6	—	—
	third	64·8	27·6	7·6	—	—
	fourth	55·2	30·8	12·8	1·2	—
20	second	66·	26·	7·6	0·4	—

There was a history of a previous attack of blackwater fever in five cases.

Case 10.—Patient stated that one month previously, he had passed blackwater for a period of two days.

Case 12.—Had an attack one year previously.

Case 15.—There were two former attacks, the first two years and the second one year previously.

Case 18.—Two former attacks, one twelve years, the other six years before the present one.

Case 19.—Had an attack one year previously.

The following kindly sent reports:—Dr. Blair Aitken, Dr. Boucher, Dr. Cobb, D.S.O., Dr. E. J. Crawford, Dr. R. P. Crawford, Dr. M. W. Fraser, Dr. Gibson, M.C., Dr. Hood Rankin, Dr. Miller, Dr. North, Dr. Pasqual and Dr. C. J. Sharp, M.C.

MALIGNANT GROWTHS.

The occurrence of malignant growths in so-called primitive races has recently aroused a considerable amount of interest. Those who believe that the apparent increase in the number of cases of cancer is due to the artificiality of the present day diet, hold that malignant disease in primitive races is rare owing to their more natural habits as regards food. Unfortunately for statistics, “canned goods” have peacefully penetrated to most parts of Nigeria, but on the other hand the refinements of the restaurant chef are not practised.

A special effort has been made to obtain as many specimens of tumours as possible and the following is an analysis of those received through the special kindness of many Medical Officers.

Squamous Cancer (Epithelioma)	of leg	2.
	of soft palate	1.
	of neck	1.
	of scalp	1.
	of jaw	1.
	of gland	1.
Adeno-carcinoma	of eye	1.
	of testicle	1.
Mucoid carcinoma	of chest	1.
	of cervix uteri	1.

The sarcomata were more numerous, numbering twenty-two but, like the carcinomata were all obtained from native Africans.

Spindle-celled sarcoma	of face	2.
	of foot	1.
	of finger	1.
	of scalp	1.
Melanotic sarcoma	of foot	4.
Lympho-sarcoma	of thorax	2.
	of jaw	1.
	of intestine	1.
Myeloid sarcoma	of fibula	1.
Glio-sarcoma	of brain	1.
Round-celled sarcoma	of humerus	2.
	of arm	1.
	of chest	1.
	of rib	1.
	of forehead	1.
	of gland	1.

The innocent or simple tumours were:—

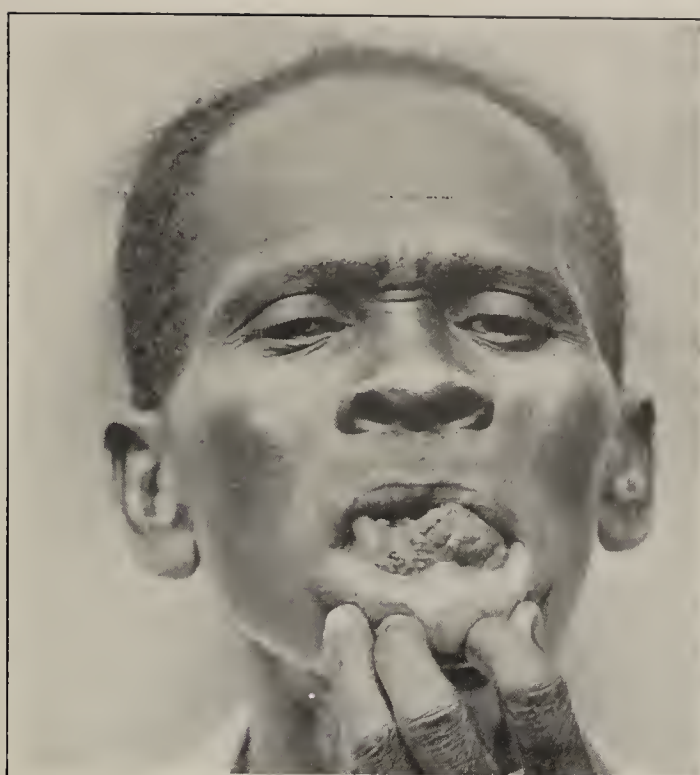
Fibroma	17.	Naevus	1.
Fibromyoma	4.	Myxoma	1.
Papilloma	3.	Adenoma	1.
Angioma	2.	Lipoma	1.
Adamantinoma	2.	Dermoid	1.

Tumours due to other causes were:—

Tubercular disease	of gland	5.
	of testicle	1.
	of epididymis	1.
	of bulbous urethra	1.
Chronic inflammatory	of gland	6.
	of testicle	2.
Gumma	of bone	1.
	of heart	1.
	of finger	1.
	of chest	1.
	of knee	1.
	of gland	1.
Onchocerca volvulus	of testicle	1.
	of chest	2.
	of scalp	2.
Elephantiasis	of thigh	1.
	of scrotum	3.
	of breast	1.
Juxta-articular nodules		3.
Goitre		3.
Retention-cyst, salivary gland,		
	with calculus	1.
Ganglion		1.
Meibomian cyst		1.
Keloid		1.



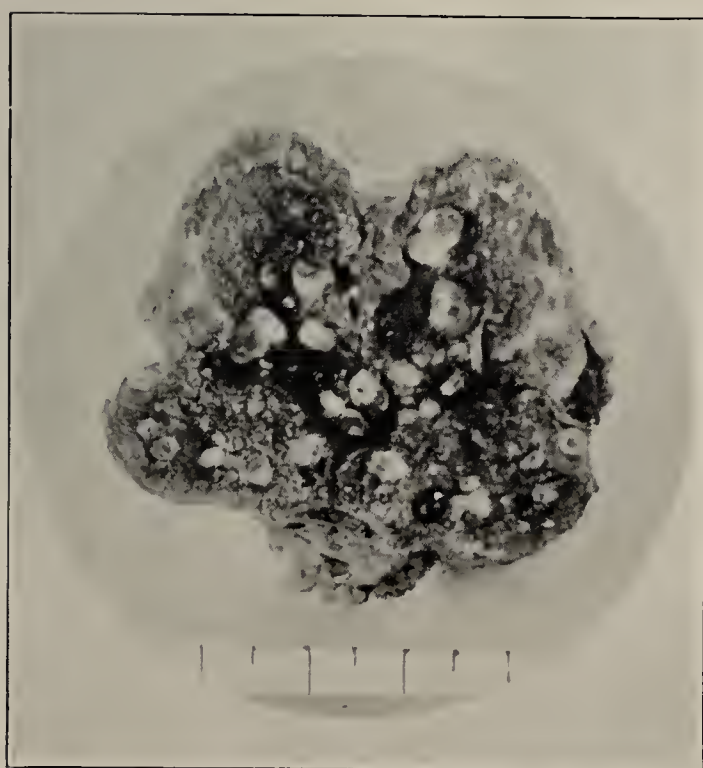
Spindle-celled Sarcoma.



Squamous Cancer.



Goundou.



Salivary gland with calculi.



Cancer of penis.

Other tissues sent for examination were the liver, spleen, kidney and other organs in cases of yellow fever, blackwater fever and relapsing fever; the lungs in several cases of pneumonia, and the brain in cases of meningitis and (in dogs) of rabies.

MISCELLANEOUS.

Sputum.—Seventeen specimens were examined. Tubercle bacilli were found in four and *B. pestis* in one.

Blood.—Nineteen blood smears were examined. *Treponema recurrentis* occurred in one, *Trypanosoma gambiense* in one and *Plasmodium malariae* in one.

The Sachs-Georgi test was done in thirty cases, the result being positive in eleven.

The Widal test was done in ten cases. The serum agglutinated *B. typhosus* in two cases, and *B. paratyphosus* *B.* in one case.

A few vaccines were prepared.

The Lagos water supply was regularly submitted to a bacteriological analysis, as were also samples from other sources.

Many smears, such as scrapings from various organs and ulcers, from discharges, from faeces and from cerebro-spinal fluid were examined.

ACKNOWLEDGMENTS.

The following kindly sent material for investigation :—

Dr. Adcock, Dr. Aitken, Dr. Anderson, Dr. Birt, Dr. Booth, Dr. Boucher, Dr. Braithwaite, Dr. Caffrey, Dr. Cauchi, Dr. Cobb, D.S.O., Dr. Courtney, Dr. Cowper, Dr. Crawford, Dr. Crichton, Dr. Dyce-Sharp, Dr. Ferguson, Dr. Forde, Dr. Fowler, Dr. Fraser, Dr. Gibson, M.C., Dr. Glover, Dr. Goodbrand, Dr. Gray, Dr. Grey, Dr. Grieve, Dr. Hogan, Dr. Jackson Moore, Dr. Kelsall, Dr. Lee, D.S.O., M.C., Dr. Mackey, Dr. Maples, Dr. Miller, Dr. Moore, Dr. Morrison, Dr. Naudi, Dr. Nelson, Dr. Newport, Dr. North, Dr. O'Keeffe, Dr. Parkinson, Dr. Pasqual, Dr. Ross, Dr. Sapara, Dr. Clive Sharp, M.C., Dr. Claude Sharp, Dr. Stephens, Dr. Stirrett, Dr. Turnbull and Dr. Wood.

PRICKLY HEAT. ITS AETIOLOGY AND PATHOLOGY.

A paper with the above title by Dr. E. C. Smith was published in the Transactions of the Royal Society of Tropical Medicine and Hygiene, Vol. XX, Nos 5 and 6, of 31st January, 1927, pp. 344-351.

The following is a somewhat condensed form of that paper. *Demonstration of the fungus in the lesions.* Scales from a desquamated area are taken and treated with caustic potash in the usual way. Any mycelial elements are readily recognised under the microscope, but in certain cases, particularly of the more chronic type, hyphae are either absent or very scanty and staining must be resorted to. In any case, staining should always be used as a confirmatory test. The simplest and most satisfactory stain is Gram.

The scales are first placed in strong (forty *per cent.*) caustic potash solution and are transferred in a few minutes to saline. They are then picked up by a platinum loop, placed on a slide, teased out into small fragments and then fixed to the slide by gentle heat.

As the horny cells have a strong affinity for carbol gentian violet, it is advisable to stain with this for only half a minute. The preparation is then treated with Gram's iodine and spirit in the usual way, dilute carbol fuchsin (one in five) for a few seconds being used as a counter stain. The preparation is then dried and examined direct with an oil-immersion lens. The mycotic bodies may be free or may occur within the epithelial cells, singly or in groups. The more usual form resembles a yeast cell in the process of budding, surrounded apparently by a capsule. The bodies vary in size, ranging from two microns in diameter to that of a red blood corpuscle. Elementary hyphae in the form of elongated buds may be seen. The yeast-like bodies show great variation in their capacity for taking up gentian violet stain. Some are an intense purple-black, others show a deep violet stippling and others again have only a capsular staining. These variations may have to do with the age of the bodies. Staining by Adamson's method gives infinitely superior results as regards mycelial elements. The budding forms when stained thus, show one or more darkly-staining dots or masses. Similar dots appear scattered through the hyphae, the whole closely resembling the conditions seen in Pityriasis.

Cultivation of the fungus.—Owing to the small amount of fungus that may be present and to the possible contamination with saprophytic organisms, treating with various strengths of alcohol can be tried but it frequently fails. It is safer to rely on numerous inoculations of suitable mediae. Four or six tubes of Sabouraud's medium can be sown with three or four fragments apiece and kept at room temperature (28.5° C.)—not incubated. The fungus should have definitely appeared within six days as an opaque, creamy-white growth. Occasionally a variety is met with which takes on a striking red colour, like that of red-lead. Such a strain is simple to detect. In the more common white or cream variety, it is advisable to pick off all suspicious-looking colonies and examine them in a drop of saline under a cover-glass, using a high dry lens with a partially closed diaphragm. In positive cases the typical yeast-like appearance of the organism is unmistakable. Frequently the fungus is mixed with cocci or sporing bacilli, so that plating out on dry agar plates and incubation at room temperature will be necessary to obtain a pure growth. Once obtained in pure culture, it can be inoculated on Sabouraud's slopes in large tubes of 1½ inches diameter. After seven days at room temperature, the fungus appears as a flat disc about 1½ centimetres in diameter, with a fairly regular edge and a dry shiny surface. In older cultures, fine striae may grow out from the edge which becomes slightly thickened. The surface, too, may show fine radial markings and also, centrally, small irregular bubbles which later give rise to pits or depressions. Some of the strains isolated grow deeply into the media producing, when cut across, a delicate fan-like appearance. The white and cream-coloured strains tend to darken with age. The red variety may become almost white but several strains of this type have retained their intensity of tint over a period of three months or more. Microscopically, the cultures are found to be composed almost entirely of budding forms, staining Gram-positive, and showing considerable variation in size. Here and there, one comes across a short mycelial filament or an elongated bud, but this is uncommon even in old cultures.

A table is given which shows the sugar reactions of the various strains isolated during the investigation.

TABLE VI.

Strain.	3,113.	1,398.	5,195.	4,891.	D.	C.	Y.	1,758.	1,450.	9,230.	8,790.
Glucose ...	+	+	+	+	+	+	0	+	+	0	+
Lactose ...	0	0	0	0	0	0	0	0	0	0	0
Mannite ...	0	0	0	0	0	0	0	0	0	0	0
Saccharose ...	+	+	0	0	0	0	0	+	0	0	0
Maltose ...	+	+	+	+	0	0	0	+	0	0	+
Dulcite ...	0	0	0	0	0	0	0	0	0	0	0
Dextrose ...	0	0	0	0	0	0	0	0	0	0	0
Galactose ...	0	0	0	0	0	0	0	0	0	0	0
Inulin ...	0	0	0	0	0	0	0	0	0	0	0
Levulose ...	0	0	0	0	0	0	0	0	0	0	?

Note.—Strains D. C. & 1,450 produce gas in glucose only. They possibly therefore belong to the Balanica group of Monilia.

Strains Y & 9,230 do not produce gas in any sugar, are red in colour and may be Monilia rosea, Zenoni.

Experimental inoculation.—Attempts were made in guinea-pigs and rabbits to reproduce the disease and test the pathogenicity of the fungus. Intra-peritoneal and intra-muscular injections and rubbing on a scarified area of a thick emulsion of a twenty-four hours' growth on agar failed, but subcutaneous injection of a similar material usually led to the formation of small abscesses at the site of inoculation, from which the fungus could be recovered, and showing when sectioned a central mass of fungal elements surrounded by a zone of inflammatory reaction.

Human inoculation was performed as follows:—An area of the skin was shaved and washed with saline. A piece of lint one inch in diameter was moistened with a thick emulsion of a twenty-four hours' agar growth, was applied to the shaven area and was kept in close contact by means of adhesive plaster. Four strains were thus tested. As a control a similar piece of lint, moistened with saline only, was applied to a similar area. The incubation period varied slightly with the strain used, but four days was the average time taken for the lesion to develop. On about the second day, however, definite pruritis was felt in the inoculated area particularly after hot drinks and exercise. By the fourth day this area was found to be covered with minute sudaminal or herpes-like vesicles, interspersed with small red papules, the whole being surrounded by an inflammatory condition of the skin. The vesicles are similar to those seen in the early stages of prickly heat but are slightly larger. The contents of the vesicles were negative both in culture and in smears, but on picking off the tops of the vesicles and examining in caustic potash solution, masses of rounded, spore-like bodies were seen, together with some budding forms and hyphae.

A day or two later the vesicles dry up and the area becomes more definitely papular in appearance. Desquamation commences about the seventh to the tenth day from inoculation. It is at first profuse, the scales coming away in large flakes: but later the process diminishes and the scales become finer and more difficult to obtain. The fungus can be demonstrated quite easily in these scales and great variety is presented as regards the size and contour of the fungal elements. When the rounded spore-like bodies are massed together a mosaic appearance results. In other groups the size of the bodies varies from that of a rather fat bacillus to the diameter of a red blood corpuscle. Numerous elongated buds and abortive attempts at hyphae-formation occur. Long branching mycelial strands are also seen many of which show lateral budding. Isolated budding forms can readily be found in the scrapings from an experimental lesion as late as six weeks after inoculation, even although the lesion has not been protected in any way. The mycelium is more prolific in the earlier stages gradually diminishing as the condition ages.

The fungus can be recovered in culture from the scales and if re-inoculated in the same manner it will again produce the lesion. If, instead of inoculating in the way described above, the culture is picked from the agar slope direct and rubbed into the unshaved skin with a platinum loop, the incubation period is greatly diminished and the lesion appears in from twenty-four to forty-eight hours.

Pathology.—On examination of sections of experimentally-produced lesions there are two features of interest (1) the presence of vesicles or pustules and (2) changes in the hair follicles.

The position of the vesicle varies. It may be so superficial as to lie entirely in the horny layer which shows a patchy hyperkeratosis and slight parakeratosis, or it may be situated deeply and encroach upon the corium, separated from it by a compressed and distorted layer of malpighian cells. Again some may show a definite relation to the sweat ducts. Others occur haphazard, frequently lying just to one side of a hair follicle. Many of the vesicles or pustules are seen to surround sweat channels as they make their way through the epidermis to the surface, their thickened, slightly hyalinised walls standing out sharply from the background of the vesicular contents. Serial sections failed to show any connection between these pustules and the lumina of the sweat channels. Here and there, however, a vesicle (or pustule) occurs showing a nodular thickening in its miniature roof. Closer examination reveals in the centre of the nodule a small opening with a hyalinised wall, and on glancing at the corium it will be noticed that the pustule is lying directly over a sweat gland. With the help of serial sections, the floor of the pustule can be shown to merge into the sweat channel arising from the subjacent gland. Whether the pustule has arisen from the sweat gland within, or whether it has merely formed around it, producing disintegration of its walls, is difficult to say. The vesicles in their early stage contain a sero-fibrinous fluid, in which lie groups of separated epithelial cells. Later the inflammatory reaction becomes more intense and the vesicle, or rather pustule is replete with polymorphs and some lymphocytes. The malpighian cells forming the sides and floor of these little excavations are oedematous and the intercellular spaces appear more distinct than normal. Many of the cells have become spindle-shaped owing to the pressure. The cell nuclei are irregularly compressed, pyknotic, and are surrounded by a clear unstained area representing the oedema fluid. Polymorph cells showing much distortion are seen in the intercellular spaces, where they have been fixed on their way to action.

With regard to the staining of the fungus in the tissues, Gram's method will pick out the spore and budding forms and will show their double contour to advantage. The mycelium, however, is stained but poorly. The same remarks apply to haematoxylin-eosin staining; with this method, the peripheral or capsular portions of the bodies take up the haematoxylin, the central parts staining pink, and the mycelium again is poorly stained mainly from a lack of contrast with the surrounding tissues. Loeffler's methylene blue, decolorised by tannic acid gives excellent results when the lesion is confined to the horny epithelium, for the fungus stains a deep blue and shows up vividly against the mauve background of keratinised cells.

The best results, however, have been obtained with alcohol-fixed sections stained by pyronin methyl green. With this stain, both the mycelium and the budding forms take on a deep, dull-red tint which contrasts sharply with the greenish-blue colour of the horny cells. The domed roofs of the vesicles are found to contain groups of fungal elements, the spore forms lying within the epithelium whilst mycelial threads insinuate themselves between the individual cells. The walls and the bases of the vesicular lesions are negative, but occasionally a fragment of mycelium that has become separated is found within the vesicle or pustule.

There are probably two factors responsible for the lesions that are produced (1), mechanical, (2), toxic.

The mechanical factor is the irritation resulting from the presence of a foreign body—the fungus in the cells—this also causing blocking of the intercellular spaces. The toxic factor also arises from the same source, *i.e.*, the fungus.

The combination of these two factors brings about a progressive oedema of the cells in the vicinity, with subsequent disintegration of those most involved resulting in vesicle formation, the vesicles later becoming filled up with polymorphs as the inflammatory reaction increases. With regard to the changes in the hair follicles, the part of the follicle involved is that which passes through the epidermis. The deeper part, or hair root is not altered nor, apparently, is the hair itself. Each affected follicle appears dilated and the cells forming the sheath through which the hairs pass are swollen, oedematous and somewhat more irregularly arranged than usual. Lying within and amongst them are clusters of the yeast-like bodies, some of which are in the budding stage, and an occasional fragment of mycelium. The yeast-like bodies are seen also to occur in an orderly line, closely applied to the issuing hair, so that the latter may be said to pass through a sleeve of fungal elements, this sleeve lying between the hair and the cells of the follicles. Groups of separated spores and hyphae also occur, as already noted, in the follicular epithelium, particularly towards its superficial extremity. Polymorphic leucocytes are seen among the cells bordering the hair follicle. The hairs and also the ducts of the glands (sebaceous) appear to be free from infection.

As well as in the roofs of the vesicles and in the hair follicles, the fungus can be demonstrated throughout the horny layer, more especially in the areas of hyperkeratosis.

In the corium the capillary vessels are distended, their endothelium is hypertrophic and many are packed with inflammatory cells, while an external layer of similar cells surrounds them. Lymphocytes are relatively more numerous here than in the pustules. Mast cells and plasma cells do not appear to take part in the inflammatory process. The sweat glands, beyond having slightly dilated lumina, do not show any notable change. The ducts, however, as they enter the epidermal layer, are here and there accompanied by inflammatory cells. Collections of similar

cells occur in connection with the deeper parts of many of the hair follicles. From the above description it is not difficult to understand why anything which causes increased sweating provokes such acutely irritating sensations in those afflicted with prickly heat. The great prevalence of prickly heat in the hot (and moist) season depends on a number of factors. In the first instance, the skin is presumably sodden by the continuous sweating so that any chance mycotic infection will gain a more certain anchorage and will have a better chance of infiltrating the softened superficial cells. The dampness of the clothes will tend to aid the propagation of any infection which they may harbour and which will, eventually become worked into the skin more easily and abundantly than in a dry state. It is a well-known fact that yeast-like bodies are occasionally found in apparently normal epithelial cells, and it is possible that the infection remains latent for long periods, breaking out only under suitable conditions. It is of considerable interest that it is in those parts which become chafed by clothes that the condition usually starts. In this respect it resembles the *Tinea versicolor* or Pityriasis of colder climes. As regards the actual source of infection, it is significant that skin lesions yielding a type of skin fungus identical to that herein described, are rife among the native African population.

SUMMARY.

A fungus showing yeast-like budding forms and branching mycelium, has been demonstrated in the epidermal scales of persons affected with the condition known as "prickly heat."

This fungus can be cultivated from the scales, and when stained appears as masses of budding yeast-like bodies which are Gram-positive. A few of the strains show short, scanty mycelial elements.

By inoculation of a culture of this fungus on human skin, a condition is produced which is comparable to prickly heat in an aggravated form. The lesion which is produced shows a primary vesiculo-papular stage and a terminal desquamatory phase. A fungus identical with that used for inoculation can be demonstrated in the scales, and obtained from them in culture.

The fungus isolated from the experimentally-produced lesion will, on reinoculation, give rise to a similar epidermal lesion.

Sections of the experimental lesion show the presence of a yeast-like fungus in the horny epithelium, in the roofs of the vesicles or pustules and in the superficial parts of the hair follicles.

CONCLUSION.

Prickly heat is a dermatitis of mycotic origin.

The causative agent belongs to the *Monilia* group.

SKIN DISEASES INVESTIGATED IN LAGOS.

Dr. E. C. Smith investigated the skin diseases in Lagos and he has written the Report which follows.

During the year, upwards of 300 patients presenting various forms of skin lesions were examined at the Out-patient Department of the African Hospital, Lagos.

It gives great pleasure to record the help received from the late Dr. W. R. Parkinson, from Dr. Keer and Dr. MacGregor and from the European Nursing Staff. Dr. Smart and Dr. Faderin, in medical charge of the police and of the prisoners, have also been most helpful. Dr. Sapara (Massey Street Dispensary) sent several interesting cases. From Dr. Macaulay (Private Practitioner) cases have also been obtained.

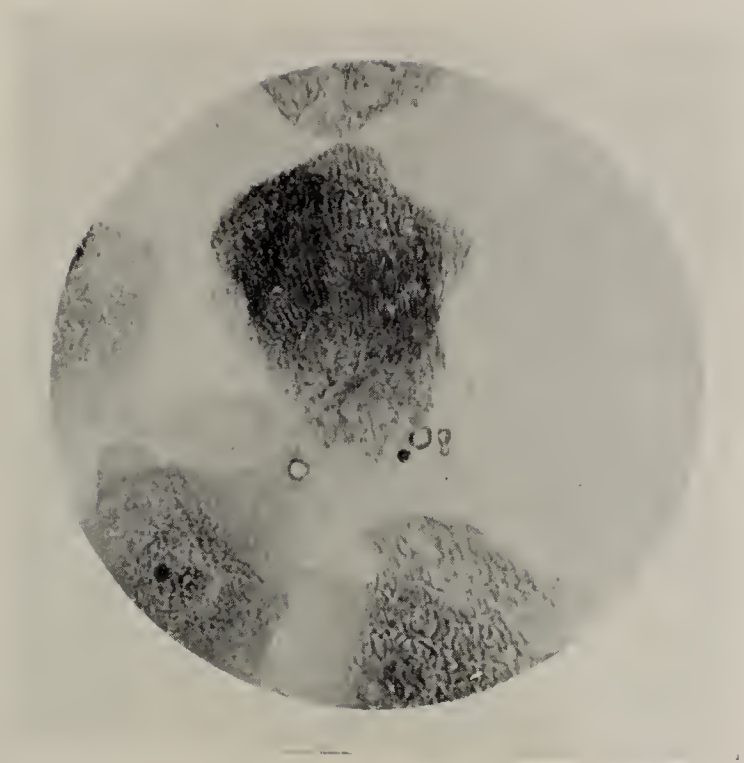


Fig. 1.

Scrapings from a case of prickly heat
showing epithelial scales and spore-
like bodies. (Gram stain $\times 850$.)



Fig. 2.

Scrapings from a more severe case; showing
clusters of " spores " with well marked
capsules. (Gram stain $\times 850$.)



Fig. 3.

Ten day culture (No. 3113) on Saboraud's maltose agar, at room temperature. The upper colony indicates faintly the fine radial processes extending into the media.

Three-quarters actual size.



Fig. 4.

The same culture, five days later, in transverse section showing the invasion of the medium by the fungus. $\times 2$.



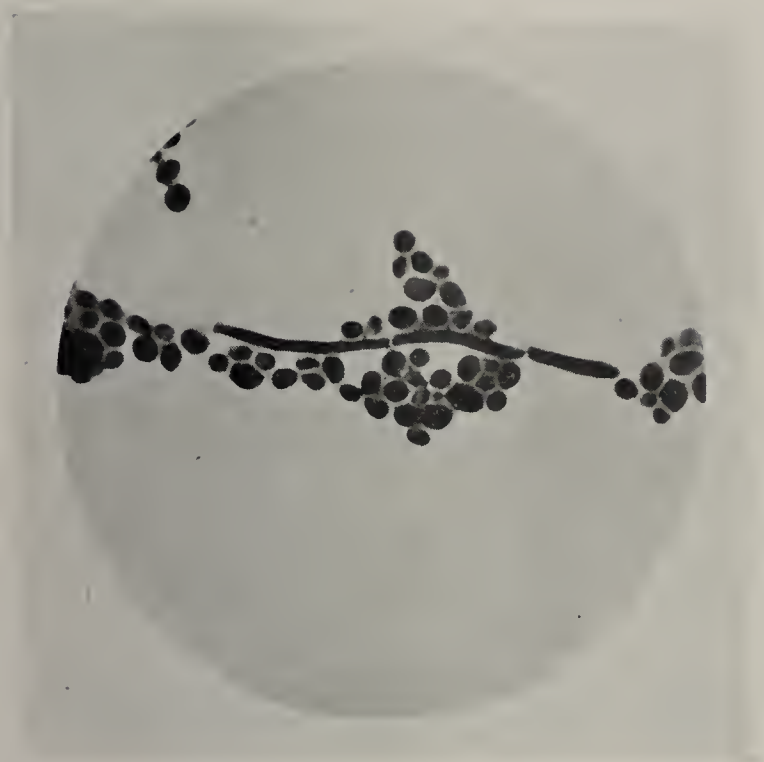
Fig. 5.

Fourteen-day culture (Strain Y) at room temperature on Saboraud's maltose agar. $\times 1\frac{1}{2}$.



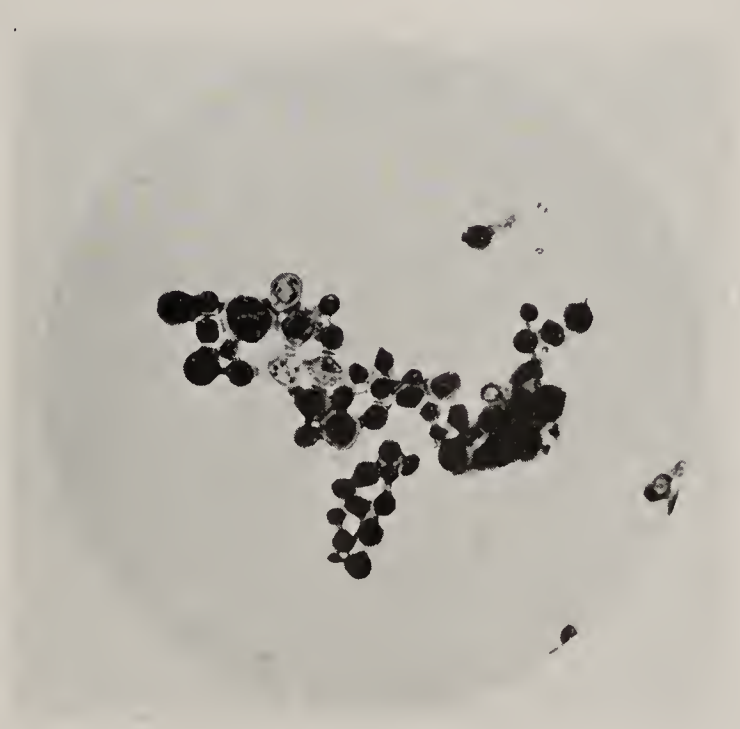
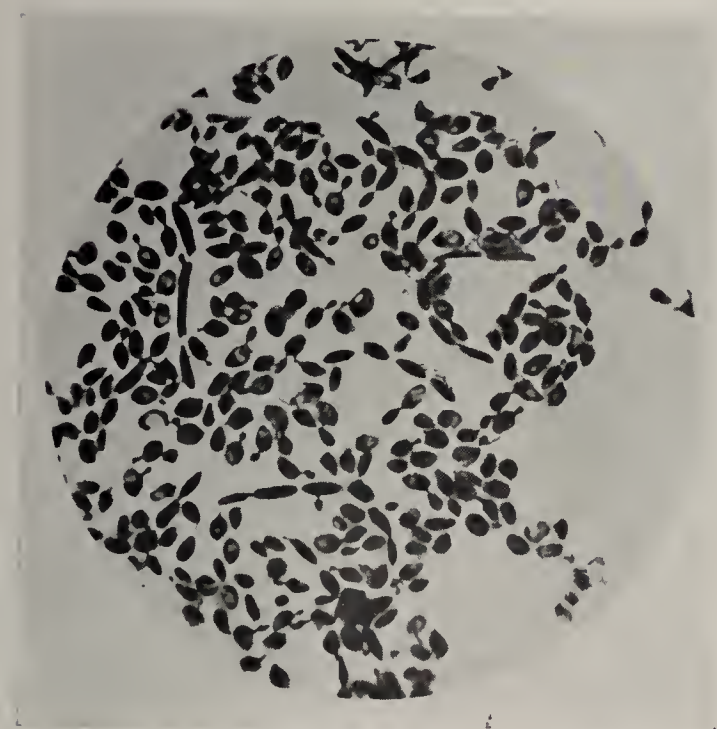
Fig. 6.

Forty-day culture of the same strain. This variety presents the red coloration described in the text. It does not invade the medium. $\times 2$.



Figs. 7 and 8.

Smears, stained by Gram, of a twenty-four-hour culture of No. 3113, showing some elementary hypha-formation. $\times 950$.



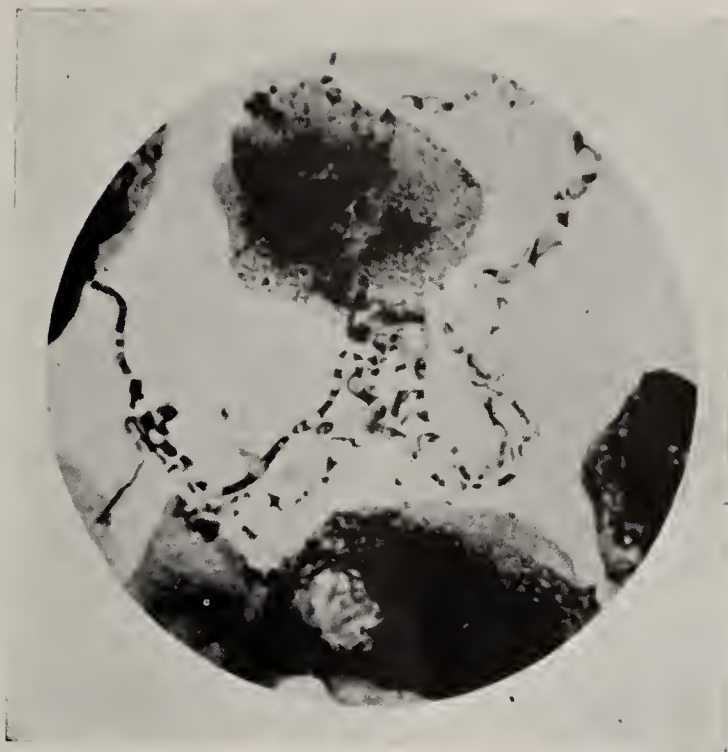
Figs. 9 and 10.

Smears made from old cultures (one to three months) showing elongated forms, hypha-formation and stippling of the spore-like bodies. Gram stain. $\times 850$.



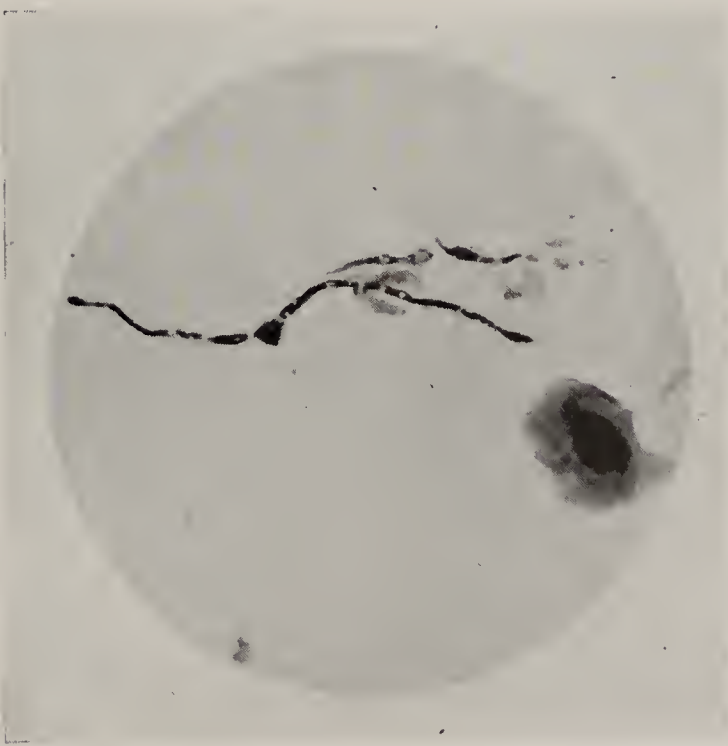
Figs. 11 and 12.

Experimentally produced patches showing
vesiculation. The technique is described
in the text.



Figs. 13 and 14.

The fungus as it appeared in the scrapings from an experimental patch. Gram stain. $\times 850$.



Figs. 15 and 16.

Well-marked mycelium in the scrapings from an experimental patch. Gram stain. $\times 850$.



Fig. 17.

Cluster of "spores" and hyphæ showing well-marked capsulation. From an experimentally produced patch nineteen days after inoculation. Gram stain. $\times 850$.

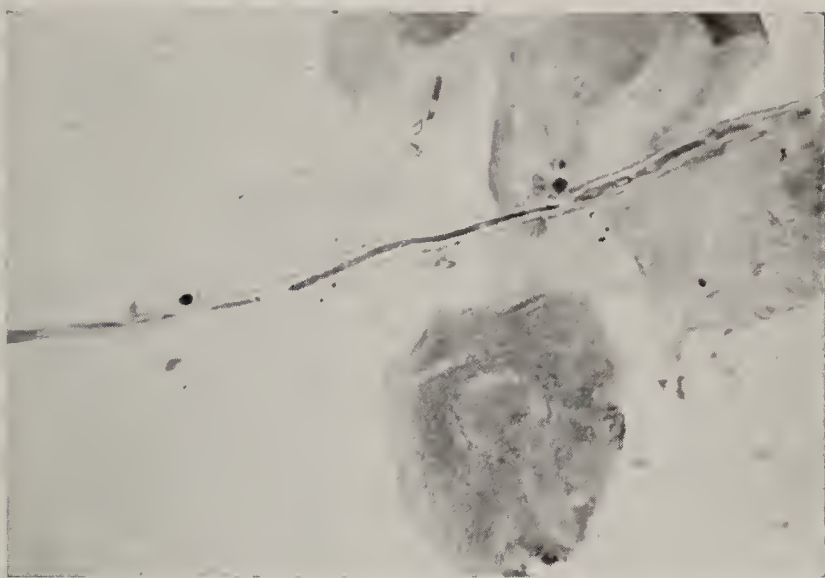
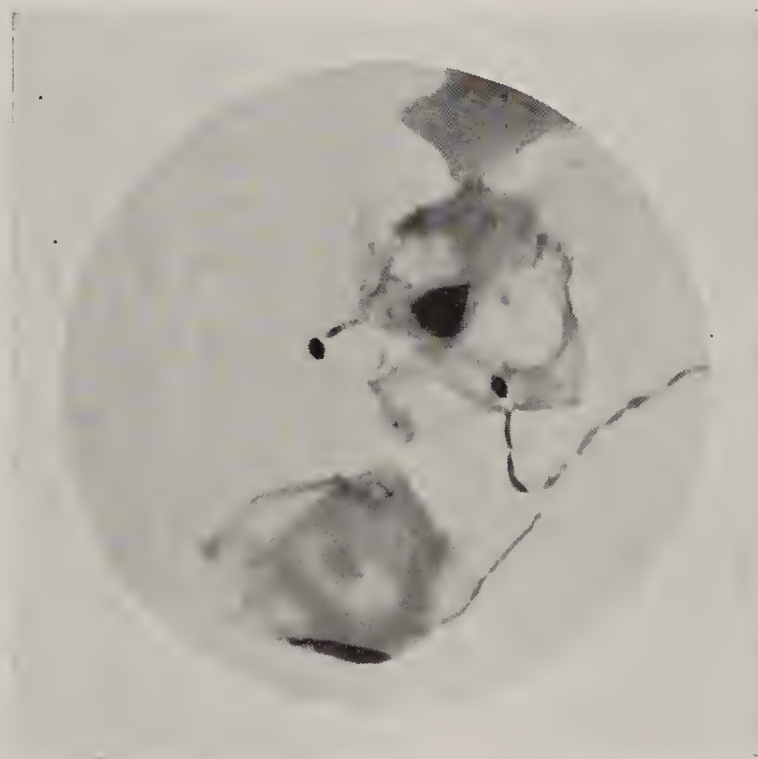


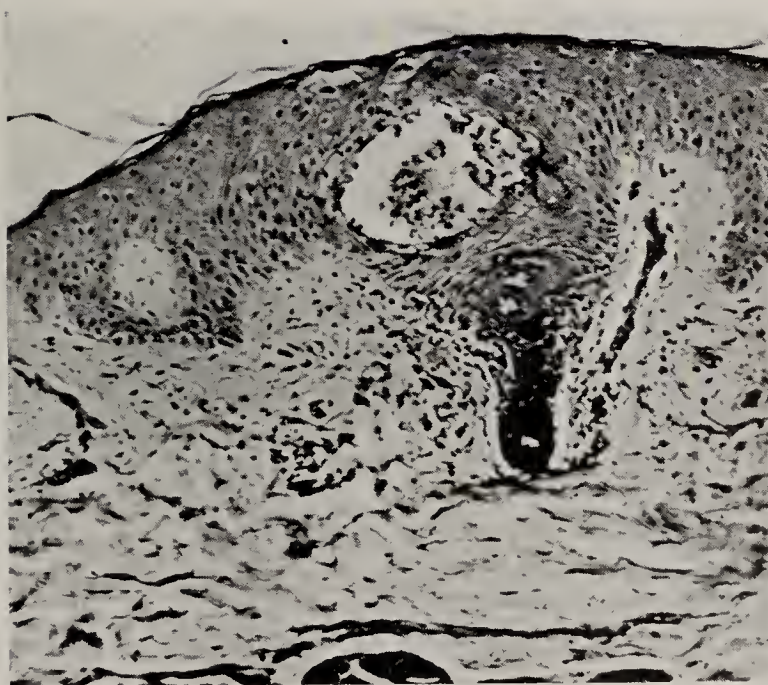
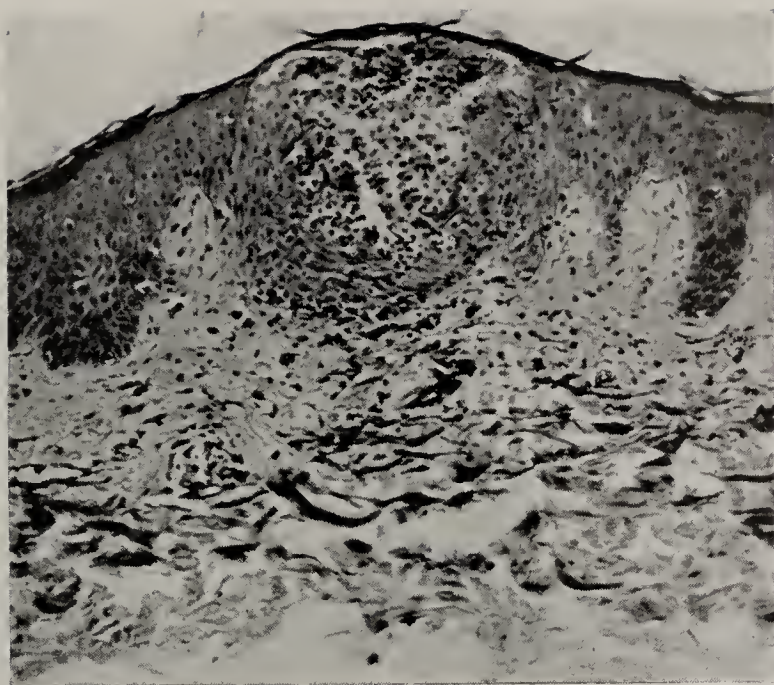
Fig. 18.

A fragment of mycelium from the same source showing lateral budding. Gram stain. $\times 850$.



Figs. 19 and 20.

Scrapings from an experimental lesion showing the close relationship between the fungus and the epithelial scales. Gram stain. $\times 850$.



Figs. 21 and 22.

Sections showing vesicle (more correctly, pustule) formation. The lower section shows a vesicle in relation to a sweat-duct.

From an experimental lesion. $\times 350$.

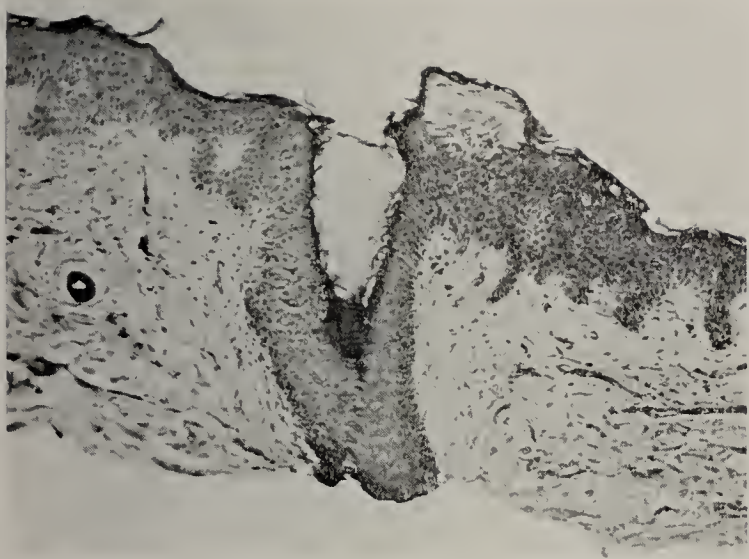


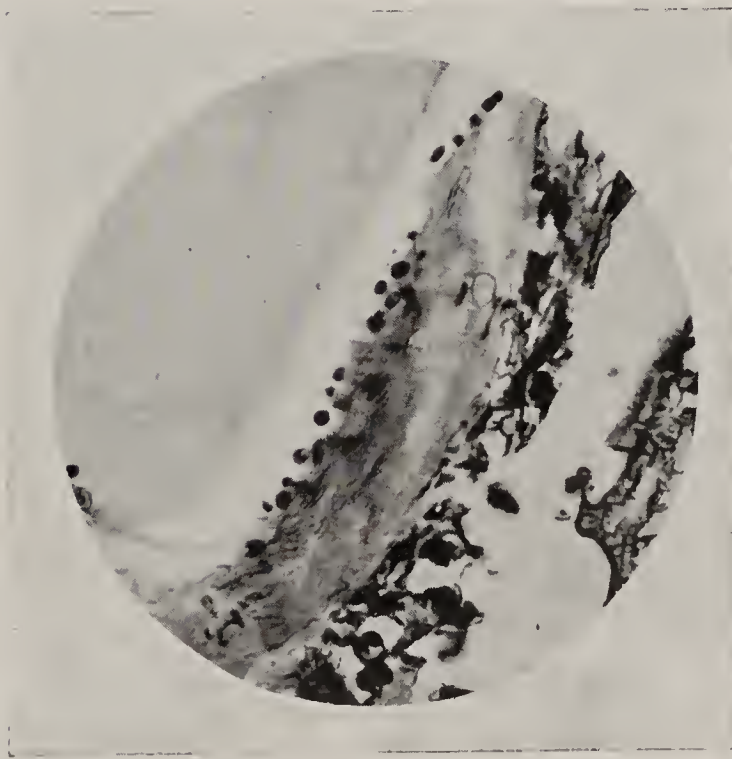
Fig. 23.

Section through a hair follicle showing its increased size and slight inflammatory infiltration of the corium. An adjacent vesicle is also apparent. $\times 90$.



Fig. 24.

Fungal elements in the wall of the follicle shown in *Fig. 23*. Hæmatoxylin-eosin stain. $\times 850$.



Figs. 25 and 26.

Sections of a hair follicle with hair *in situ* showing the position occupied by the fungus. Hæmatoxylin-eosin stain. $\times 850$.

(Microphotographs by Mr. F. W. RANDOLL, Laboratory Attendant).

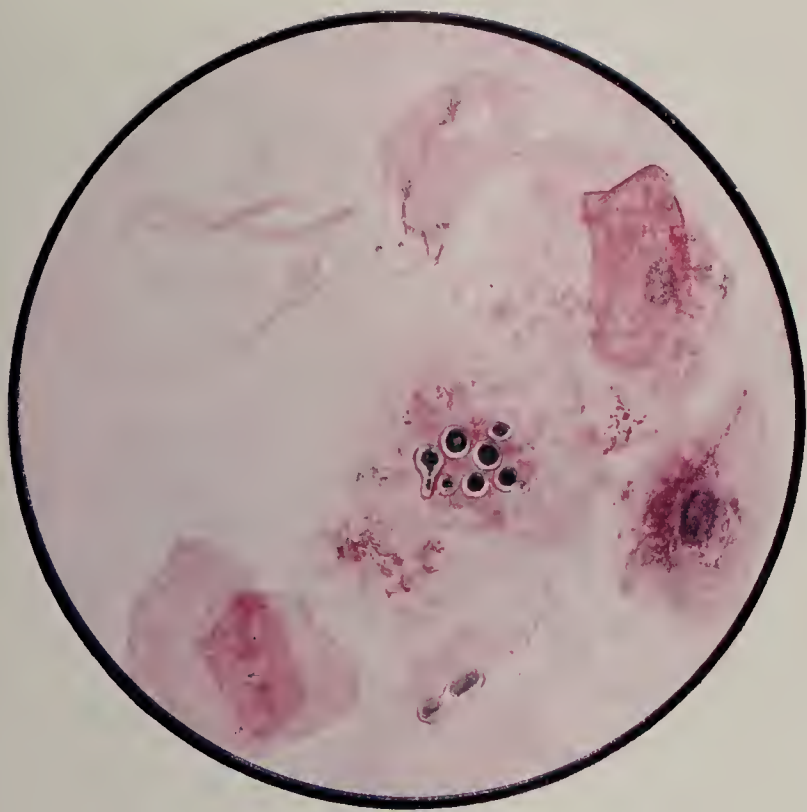


Fig. 27.

Drawing of a typical group of spore-like elements as seen in the scrapings from a case of prickly heat when stained by Gram. $\times 850$.

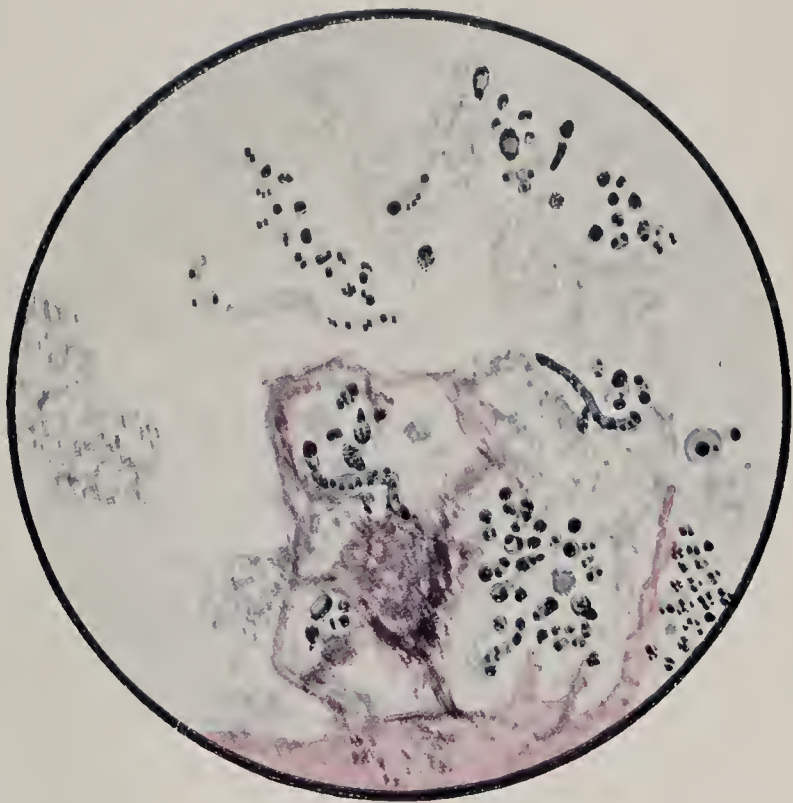


Fig. 28.

The fungus as it appears in the shaved off vesicular tops of an experimentally produced lesion. Staining by Adamson's method. $\times 850$.

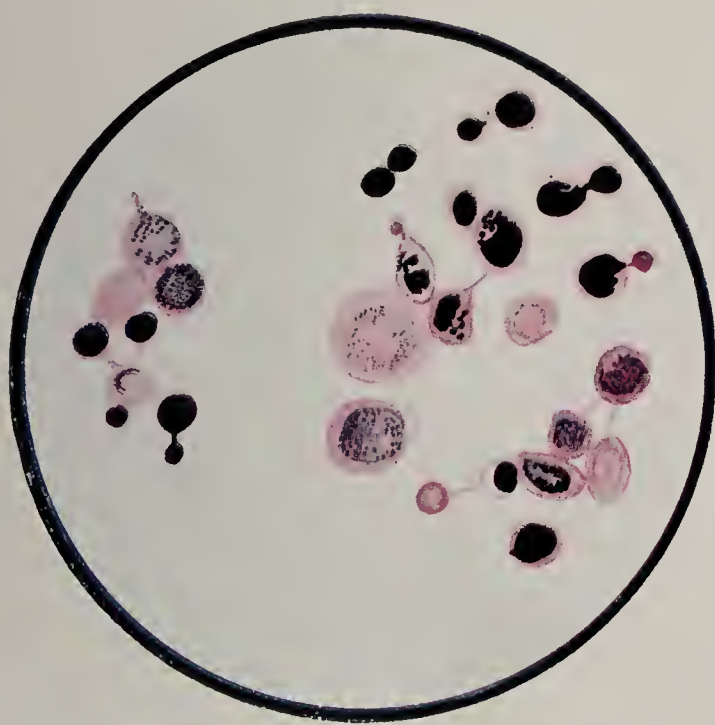


Fig. 29.

Drawing of a smear of a twenty-one day old culture, grown at room temperature on ordinary agar showing bizarre staining effects, and great irregularity in the size of the yeast-like bodies. Gram stain. $\times 950$.

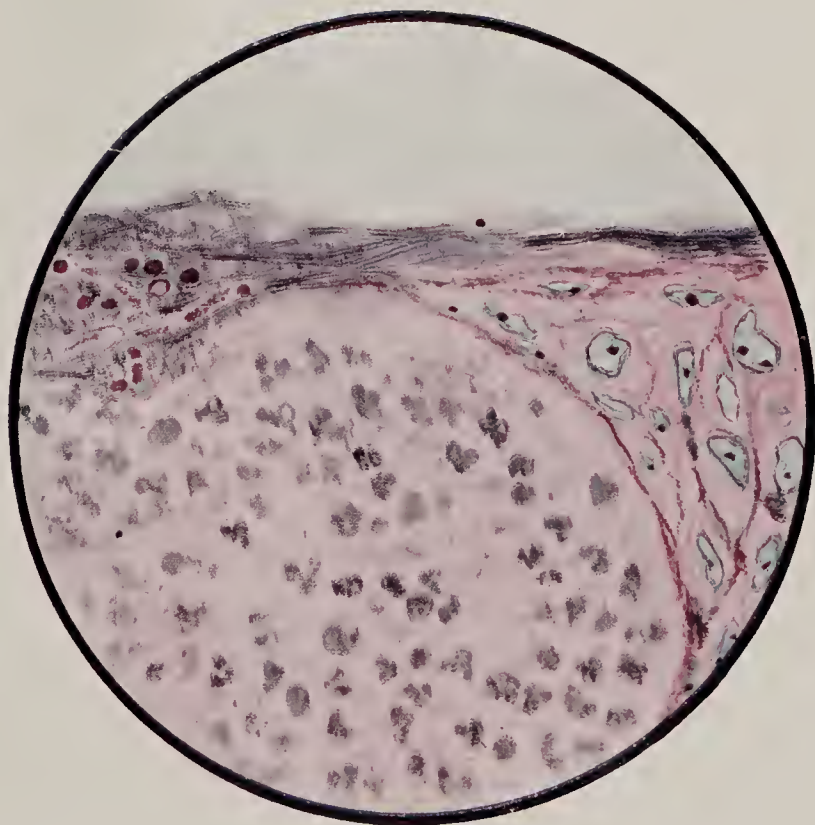


Fig. 30.

Drawing of a section through portion of a vesicle showing mycelium and "spores" in the epithelial cells forming its roof. Alcohol fixation. Pyronin Methyl Green stain. From an experimental lesion. $\times 850$.

As mentioned in the last Report, the method of examination is, wherever possible, threefold :—(I) Microscopical (examination of smears and scrapings); (II) Cultural; (III) Histological (a biopsy is made and the tissue so obtained is sectioned). In many cases, the last method is found to be the most reliable and helpful.

Photographs of the new or more interesting lesions are taken and a clinical record of each case is kept.

The following synopsis will give some idea of the variety of lesions.

	Number of Cases.
Cancer (squamous) (leg ulcer, tumour penis, papilloma back)	3.
Dermatitis papillaris capillitii (sycosis nuchae)	3.
Favus (Achorion Schonleinei)	1.
Herpes Zoster	4.
Kerion (suppurative tinea of the scalp) ...	4.
Lepothrix or trichomycosis	numerous.
Leprosy (one nodular, two anaesthetic) ...	3.
Lichen pilaris	8.
Madura mycosis	2.
Dermal moniliasis	36.
A condition clinically resembling " Mossy Foot "	3.
Onchocerca volvulus (tumour on scalp, tumour on thigh)	2.
Psoriasis (non-syphilitic)	1.
Impetiginous eruptions	10.
Sycosis barbae	2.
Syphilitic ulcers	4.
Simple tropical ulcer	65.
Tinea capitis (varieties not yet classified) ...	20.
Tinea of other regions (unclassified)	51.
Tubercular ulcers	3.
Varicella	4.
Yaws (diagnosis made only when the causative agent found)	7.

A considerable number of cases have been omitted from the above list. This is due to their causation being more or less obscure. It is hoped that as more cases of a similar type are examined, a higher diagnostic percentage will be obtained.

Apart from the above, numerous cases were examined at Port Harcourt, during a brief visit there. Many examples of untreated yaws were seen and the causative agent obtained in smears and sections. A few examples of the condition known as Gangosa were also seen, but as they were under treatment no extensive examination was made.

Leprosy was seen in all its varieties, one case in particular being worthy of mention. The patient, a male adult presented himself with a diffuse papulo-pustular dermatitis, and complaining of pain in the nose. Nasal smears were rich in acid-fast bacilli, as were also the contents of the minute pustules. The organism was also demonstrated in sections of a pustule. Apart from a general dryness of the skin and the nasal pain there was nothing to suggest leprosy, the condition resembling a septic dermatitis. It is interesting to note that leprosy can assume this form.

One case of scabies was diagnosed by finding the parasite and one case of Molluscum contagiosum was noted in a child.

That such an interesting number of cases were seen in a short time (one week) at Port Harcourt was due to the kindness of Dr. H. R. M. Ferguson.

A condition known as "Mossy Foot" has been alluded to. Though bearing a certain resemblance to the "Mossy Foot" of American writers, there is sufficient divergence to show that it must belong to a different group. The lesions, as seen in Lagos, commence as very dry verrucose patches confined to the dorsum of the feet. Later, papules or nodules form which are still covered by the papillated skin. Sections show a vascular granulation tissue, covered by the well-marked papillary epithelial layer and containing numerous infiltrative areas, in some of which giant-cell formation has occurred. Examination for fungi, spirochaetes or acid-fast bacilli have so far been negative. Some of the lesions are not unlike those of *Ichthyosis hystrix* as shown in standard textbooks.

Impetiginous eruptions have been put down as ten in number. This must not be taken as representing a true percentage, as pustular eruptions on the hands are exceedingly common and many seem to have originated in the interdigital clefts. Repeated examinations have as yet failed to reveal parasites (*i.e.*, scabies). Nor do the lesions show any attempt at burrow formation, which is so diagnostic in European cases. Cultures and smears have consistently yielded staphylococci, but it would seem advisable to continue the investigation of these cases before placing them in a septic category.

Several excellent examples of Bockhart's impetigo were met with. Both cases of Madura mycosis were obtained through the kindness of Dr. Gray and Dr. Aitken. The first was a young Syrian girl who presented the typical book-picture of madura-foot. The second case, an adult male native of Lagos, showed lesions which were most atypical. The condition involved the leg and was manifested in the form of indurated areas situated deep to and involving the epidermis. Some of these areas were capped by scabs which, on removal, revealed a minute raw area with sometimes a bead of pus. One such lesion admitted the passage of a probe for several inches. When the areas were opened into, dense scar-like tissue was exposed. At one place the tissue was softer and could be readily scraped away. It is important to emphasise the difficulty which may arise in the diagnosis of such cases. In the first patient, although numerous smears were made, in only one was the fungus recognised.

Smears from the second case revealed nothing beyond the common pyogenic organisms.

In both cases, although eight to twelve blocks of tissue were prepared from each, in only one block from each set was the characteristic fungal mass found. Grains or granules were never noted in the second case. In the first, they were found during the amputation of the foot when a large amount of necrotic bone was removed. Several large yellow grains were then picked out and cultured. The cultures, impure from the first, were eventually over-grown by the secondary organisms. Cultures from the second case were entirely negative.

SIMPLE TROPICAL ULCER AND/OR TROPICAL SLOUGHING PHAGEDENA.

This condition received considerable attention, and the results obtained from the examination of a large number of cases are now given in a condensed form. Fifty *per cent.* of the cases occurred in males. Mostly these were young adults. All the ulcers were in the region of the lower limbs—an important point in etiology—and sixty-two *per cent.* were in the region of the malleoli, the area most exposed to injury when the avocations of life are carried on in bare legs. Almost all the cases gave a history of traumatism, mostly by sharp sticks. The condition is almost invariably described as commencing like a boil or pimple which becomes scratched, then ulcerated, and then rapidly extends. The shape and the extent of

the ulcers vary greatly, according to their duration; and the term "sloughing tropical phagedena" is an apt one, as the majority of them are covered with a foul purulent layer (the result of neglect or of filthy wrappings) which when cleaned away leaves a raw base, composed of granulation tissue and surrounded by a raised, indurated edge. Later, the ulcer may extend deeply and expose muscle, tendon and bone. Neighbouring glands usually show but slight involvement, as the condition is primarily a local one. As regards the etiology of these ulcers, various causative factors have been put forward from time to time. Chief among these are:—

I.—Syphilis.

II.—Yaws.

III.—*Spironema schaudinii* (Prowazek) in symbiosis with fusiform bacilli of Vincent's type.

IV.—Some unknown bacterial agent acting in connection with malnutrition and injury.

Both syphilis and yaws can produce ulcerated lesions and there is always the possibility of such a condition forming the starting point of the so-called "ulcus tropicum." Apart from the possibility of such a coincidence, however, there is no etiological relationship. In support of this view, the following facts are mentioned:—

- (a) The spirochaetes of syphilis or yaws have not been found in smears made from the ulcers in Lagos, when examined by darkground and by Fontana's method of staining.
- (b) Many of the patients were young children. They presented none of the lesions characteristic of congenital syphilis.
- (c) In fifteen typical cases (under the supervision of the late Dr. Parkinson), full courses of N.A.B. (four to six injections of 0.6 gramme each) were administered. No improvement took place, the ulcer either remaining stationary or definitely retrogressing.
- (d) A large majority of the cases, when kept at rest and treated with simple dressings (saline, Balsam of Peru, hot stupes) rapidly cleared up.

It remains to show whether the *Spironema schaudinii* (Prowazek) or another organism is the etiological factor.

Reference must first be made to some experimental work which was carried out in connection with this type of ulceration.

Adult guinea-pigs were inoculated subcutaneously with amounts varying from one to two c.c. of an emulsion made from such ulcers in saline. In some of the animals death occurred within twenty-four hours. Post-mortem examination revealed a severe broncho-pneumonia, and sometimes also, splenic abscesses occurred. In such animals, cultures from the heart, lungs and spleen all yielded a profuse growth of a small, Gram-negative bacillus, which was motile. The condition, therefore, was a septicaemic one. Other guinea-pigs showed merely a generalised congestive condition with here and there minute haemorrhages, cultures from the various organs being negative. At the site of inoculation, however, an oedematous, green-stained area is usually found, which when incised exudes a thin greenish fluid rich in Gram-negative motile organisms readily obtained in culture. These animals apparently die from a toxæmia, the organisms remaining localised. Again, in a certain number of animals, a rapid rise of temperature (103° to 105° F.) occurred on the day following the injection. The tempera-

ture rapidly subsided and about the fourth to the sixth day the site of injection was marked by a large abscess, giving rise later to a spreading ulcer which eventually healed within a few weeks. (See table of inoculation experiments). Smears from these abscesses and from the resultant ulcers showed large numbers of motile Gram-negative bacilli, many of them lying within polymorph cells. The organism at times assumed a diplococcal form. Cultures were easily obtained and they frequently developed an intense bluish-green colour, later changing to brown (*Bacillus pyocyaneus*). If the contents of one of the experimental abscesses were aseptically aspirated (*i.e.*, before the abscess had burst), and afterwards injected subcutaneously into other guinea-pigs, in quantities varying from 0.5 to 2 c.c. exactly similar results were reproduced. The contents of the abscesses were repeatedly examined by the dark-ground method but no spirochaetes were found. Cultures and smears made from the ulcers in man and taken after thorough cleansing with saline, yielded an organism similar to that obtained from the experimental lesions. When obtained in pure culture and inoculated into guinea-pigs, identical results were obtained, as above.

Though many of the cases healed up completely with simple remedies, particularly saline and Balsam of Peru, there were a certain number which resisted all treatments or healed up only to break down again. In some of these cases marked improvement was obtained by autogenous vaccine treatment. It is possible of course, that such amelioration could be explained on the principle of "Protein shock." An interesting point, however, was brought out by the vaccine treatment. It so happened that on several occasions a case treated by a vaccine had healed, the patient's serum agglutinating his own strain of *B. pyocyaneus* up to one in 250 or higher. Then a freshly broken-down area would appear and spread rapidly over the whole original ulcer site. A strain of *B. pyocyaneus*, not agglutinated by the patient's serum could be obtained from this area. A fresh vaccine made from this second organism would produce healing and endow the patient's serum with new agglutinating properties. Evidently, then, in some cases a re-infection may occur with a different strain of *B. pyocyaneus*.

From a few of the most stubborn cases, a pure culture of a diphtheroid was obtained, but such cases were unusual.

It has been claimed that cures of these ulcers have been obtained by prolonged N.A.B. treatment. Such statements are apt to be misleading, because, as has already been seen, ulcers of a syphilitic origin are in the minority in Lagos. Again, when N.A.B. treatment has been given over a long period, it is difficult to know whether the improvement has been effected by its specific or by its general tonic action. In any case, such patients are being treated either as indoor or outdoor, so that local treatment in the form of clean dressings is being provided, which in itself greatly conduces to recovery.

Autopsies were made on several cases of simple tropical ulcer. In only one case, however, was the time interval sufficiently short to exclude the possibility of post-mortem infection. This case is described below.

Case J.Z. Male. Age eighteen years. Admitted to African Hospital 29th March, 1926.

Died 6th August, 1926.

Autopsy made within one hour of death.

History.—On admission the case presented an irregularly ulcerated area on the posterior and lateral aspects of the left leg in its lower third just above the malleoli. He was treated with Eusol dressings and Pot. Permanganate baths. Slight improvement

followed. A vaccine was made and six doses 0.5 up to 4 c.c. were administered at intervals of four days. This produced a marked temporary improvement, but the ulcer broke down again within a few weeks and the patient began to run an irregular temperature between 99° and 101° F. A new vaccine was started on 17th May, 1926, but had no effect, the ulcer spreading and exposing a portion of the Tendo achilles. Cultures gave a pure growth of *B. pyocyaneus*. A third vaccine was made and large doses given (1, 2, 4 and 6 c.c.). This coincided with much improvement in the general condition of the patient, the febrile temperature subsided and he became more cheerful. A month later the ulcer broke down again and commenced to undermine the surrounding skin. The patient was now given six doses of N.A.B. of 0.6 gramme each. No improvement was noticed, he became gradually weaker and again exhibited an irregular temperature. Compounds of bismuth and mercury were also used in the treatment but were of no avail. The patient died a month later.

Post-mortem.—Marked emaciation. Abdominal fat very scanty. Skin dry and atrophic.

Abdomen. Large intestine distended with gas. No excess fluid in peritoneal cavity. No peritonitis.

Small intestine slightly inflamed in its middle portion.

Small soft glands were scattered all through the gastro-colic omentum and the mesentery.

Kidneys normal in size, somewhat tense. Opaque on section.

Cortex relatively wide. Vessels distended. Capsule strips easily.

Liver. Large and cut surface dull and slightly granular in appearance. Negative for amyloid change.

Stomach contains green-stained fluid. No apparent inflammation.

Spleen small and firm.

Appendix, pancreas and suprarenals normal although the last was pale in colour.

Thorax. Heart. Valves normal. Aorta perfect. Muscle tissue dark and rather soft.

Lungs. No pleural adhesions. Substance of the organs very pale, collapsed, tough and doughy in consistence.

Small darkly-mottled lymph glands in both groins.

Cultures made on Agar and in broth from—

Heart blood.

Spleen.

Kidneys.

Liver.

Glands in groins.

Glands in the mesentery.

All were negative.

Smears were also made from the organs mentioned. No bacteria or spirochaetes were found in them when stained.

Sections were made from all the organs. The only findings worthy of record are those in the sections stained for fat by Sudan III.

Heart.—Cells showed well marked staining granules at each nuclear pole.

The condition was one of “Brown atrophy.”

Liver.—Diffuse fatty degeneration involving the whole lobule and merging peripherally into blotches of fatty infiltration.

Kidneys.—Faint traces of fatty degeneration.

From these findings it would appear that the condition was a toxæmic, not a septicæmic one.

It has to be recorded that the smears from nearly all the ulcers examined showed the *Spirochaeta schaudinii* and the fusiform bacilli of Vincent's type. In sections, too, these organisms may be found in large numbers, at times forming a narrow band just deep to the surface of the ulcer. They are admirably demonstrated by Giemsa staining. In view of their almost constant presence, it is possible that they are a factor, either in the causation or in the persistence of the ulcers, but if that be so, it seems strange that the lesions do not yield to Salvarsan or to its allied compounds. Conditions in the mouth, such as Vincent's angina, produced by an apparently identical organism, heal rapidly under treatment with Salvarsan.

With reference to the role which *B. pyocyaneus* plays in connection with *ulcus tropicum*, the question of the production of agglutinins to that organism in the patient's blood arises. It has to be recorded that in the present series, there was only one case in which a positive agglutination was obtained with the serum of the patient (one in fifty), apart from vaccine administration.

The organism, however, may act by producing a toxæmia in the majority of cases, only becoming generalised and causing a septicæmia in isolated instances. It is interesting to note that a bacteriophage, active for *B. pyocyaneus* was obtained from several of the cases under investigation. Pure cultures of the spirochaetes and the fusiform bacilli were obtained in hydrocele-glucose-agar broth under anaerobic conditions but no results followed animal inoculations (guinea-pigs) of such growths.

The problem may be resolved into the following parts:—

- I.—Are the spirochaetes (*Sp. schaudinii*, Prowazek) alone responsible for the disease or are they merely saprophytic?
- II.—Is *Bacillus pyocyaneus* alone responsible or is it merely a contaminating agent?
- III.—Do the spirochaetes referred to act in symbiosis with *B. pyocyaneus*, or even with other organisms in order to bring about the condition?

Taking into consideration the results of investigation detailed above, along with the history, the site, and the frequent complete healing with simple dressings, it is suggested that *Bacillus pyocyaneus* is a possible causative organism, aided by malnutrition and fatigue on the part of the patient.

Summary.—Syphilis and yaws have no relation to “Tropical sloughing phagedæna” (*ulcus tropicum*).

Strains of *Bacillus pyocyaneus* of considerable virulence have been isolated from numerous cases of *ulcus tropicum*, and have produced similar ulcers on experimental inoculation into guinea-pigs.

The spirochaetes obtained by culture from the ulcers have yielded no experimental results.

SOME EXPERIMENTAL RESULTS OBTAINED IN GUINEA-PIGS.

G.P. No.	Material used.	Temperature.	Result.	Cultures.	Smears.	Remarks.
1.	Saline emulsion of material from case of Ulcus tropicum. Dark-ground shows masses of spirochaetes and fusiform bacilli. Subcutaneous inoculation, in two places, $\frac{1}{2}$ cc. each.	2nd day, 104.5° F. 3rd day, 103.4° F. 5th day, 102° F.	Two days after inoculation areas were gangrenous. Slight greenish discharge. Six days after inoculation, definite ulceration, about 1 cm. in diameter. Thick white discharge. Ten days later, ulcer began to heal.	Gram-negative, motile bacillus.	Many Gram-negative bacilli. No spirochaetes or fusiform bacilli seen.	Dark-ground examination of the discharge in saline showed no spirochaetes or fusiform bacilli.
2.	Pus from an ulcer tracking up leg. Emulsion in saline, $\frac{1}{2}$ cc. inoculated subcutaneously, and $\frac{1}{2}$ cc. injected into testis.	2nd day, 105.1° F. 3rd day, 103.6° F. 5th day, 102.4° F.	Small ulcer at site of subcutaneous inoculation on 6th day. Purulent discharge. Testis showed a temporary swelling. On 7th day, ulcer began to heal.	B. pyocyaneus is present.	Small fat Gram-negative bacilli.	Dark-ground examination negative for spirochaetes.
3.	Same emulsion as No. 2. 2 cc. injected subcutaneously in the abdominal region.		Died next day. Large gangrenous area corresponding to site of injection.	B. pyocyaneus is present.		Juice from organs negative for spirochaetes. A few found in fluid from gangrenous area.
4.	Emulsion in broth of material from an ulcer. Dark-ground shows abundant spirochaetes and fusiform bacilli. $\frac{1}{2}$ cc. injected subcutaneously.	2nd day, 104° F. 5th day, 102° F.	5th day, indurated, inflamed, swollen area with soft fluctuating centre. Ulcer formed 9th day. On 17th day, healing nearly complete.	B. pyocyaneus is present.	Gram-negative bacilli numerous.	Dark-ground examination of discharge negative for spirochaetes and fusiform bacilli.
5.	Broth culture (48 hours) of a Gram-negative bacillus isolated from a case of simple tropical ulcer. $\frac{1}{2}$ cc. injected subcutaneously. This strain developed a well-marked blue colour on culture.	2nd day, 104.5° F. 3rd day, 103.6° F. 5th day, 101° F.	4th day, indurated, inflamed area. 5th day, fluctuation present. 6th day, ulcer formed, thick purulent discharge. 20th day, healing well advanced.	Pure culture of a Gram-negative bacillus, both before and after ulceration.	Gram-negative bacilli, many intracellular.	
6.	1 cc. same material as in No. 5 used injected subcutaneously.	2nd day, 105° F. 3rd day, 100° F.	3rd day, animal dead. Skin of abdomen oedematous and greenish in colour.	B. pyocyaneus in organs and exudates. All gave marked coloration.	Gram-negative bacilli numerous.	P.M. Small abscesses at site of inoculation, with zone of inflammation.
7.	$\frac{1}{2}$ cc. 48 hour broth culture of Gram-negative motile bacillus obtained in pure culture from a case of simple tropical ulcer. This strain never produced any coloration on culture. The inoculation was made subcutaneously in the abdominal region.	2nd day, 104° F. 3rd day, 105° F. 4th day, 103.5° F. 7th day, 101.5° F.	4th day, indurated area. 6th day, fluctuation present. 7th day, small ulcer formed and remained discharging until 10th day, when healing commenced.	Pure cultures of a Gram-negative bacillus from the discharge.	Gram-negative bacilli numerous.	Bacillus used would be grouped as pyocyanoid. Many such were isolated, identical with B. pyocyaneus except its inability to produce pyocyanin.
8.	Subcutaneous inoculation. 2 cc. broth culture, same organism as was used in No. 7.	2nd day, 104° F. 3rd day, 103° F. 4th day, 100.5° F.	Inoculated area gangrenous and oedematous on 3rd day, dead on 4th day.	Positive cultures organs and exudates.	Gram-negative bacilli numerous.	Sections of organs showed Gram-negative bacilli.
9.	1 $\frac{1}{2}$ cc. 48 hour broth culture of a strain of B. pyocyaneus isolated from a case of ulcus tropicum. Subcutaneous inoculation.	2nd day, 105° F. 3rd day, 104° F. 4th day, 102.5° F. 6th day, 101° F.	7th day, large irregular ulcer, edges indurated and undermined in places. Thick pus obtained on squeezing. 13th day, ulcer commenced to heal.	B. pyocyaneus in pure culture from pus.	Gram-negative bacilli numerous.	
10.	2 cc. 48 hour broth culture, strain of B. pyocyaneus from case of tropical ulcer.	2nd day, 105.8° F. 3rd day, 105° F. 4th day, 104.5° F. 5th day, 102° F.	Animal very ill on 4th day, muscular twitching. Died on 5th day.	Positive cultures from spleen and heart.	Gram-negative bacilli numerous.	Sections of spleen showed clumps of Gram-negative bacilli in the abscess areas.



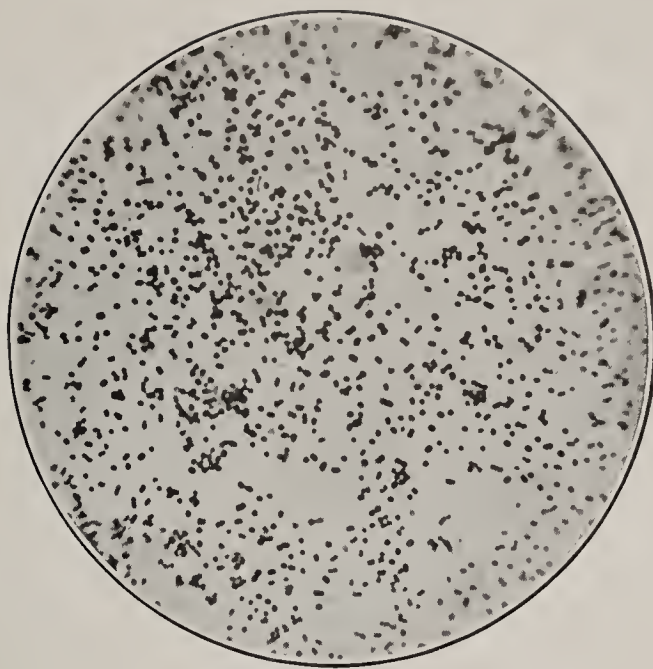
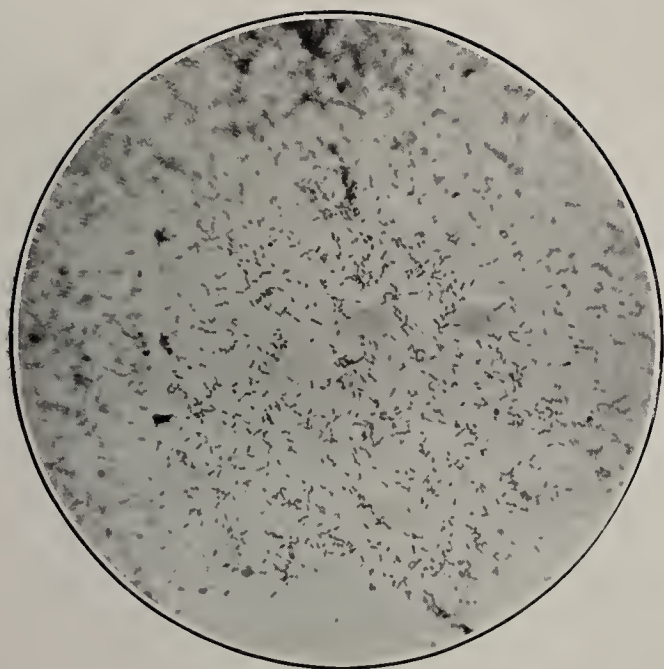
EXAMPLES OF TROPICAL ULCER.



EXAMPLES OF TROPICAL ULCER.



EXAMPLES OF TROPICAL ULCER.



B. PYOCYANEUS FROM CULTURE.



EXPERIMENTAL TROPICAL ULCER IN GUINEA-PIGS.





EPITHELIOMA CONTAGIOSUM.

The naked eye appearances, post-mortem, in the guinea-pigs which died as the result of inoculation were as follows:—

G.P. No. 3.—Scattered peritoneal haemorrhages, forming fused areas at region of injection. Fibrinous exudate present in peritoneal cavity. Lungs very congested. Heart soft and flabby.

G.P. No. 6.—Small abscess at site of inoculation, surrounded by a zone of inflammation. Lungs show numerous minute abscesses, surrounded by intensely congested zones. Liver pale and swollen. Spleen swollen and very congested. Haemorrhagic areas in abdominal muscles.

G.P. No. 8.—Marked oedema of abdominal and thoracic muscles. Haemorrhages and small abscess areas in the peritoneum. Omentum congested and showing numerous abscesses, densely adherent to small intestine and to anterior abdominal wall, at site of inoculation. Liver shows multiple minute abscesses. Lungs are congested and show haemorrhages and abscess formation. Kidneys show minute subcapsular haemorrhages. Stomach and alimentary canal acutely inflamed.

G.P. No. 10.—Spleen markedly enlarged, with irregular abscess areas similar in appearance to infarcts. Liver enlarged, pale and cloudy. Kidneys show no apparent change. Lungs show marked patchy congestion.

EPITHELIOMA CONTAGIOSUM.

Epithelioma contagiosum (Geflügelpocke). A small epidemic of this interesting condition occurred in Lagos. In all ten chickens were fatally involved. The typical crusted protuberances were present about the eyes, beak and comb of the birds (*see* photographs). Sections showed the presence of the inclusion bodies in the protoplasm of the epithelial cells. Inoculations into pigeons and rabbits proved negative.

A PATHOLOGICAL SYNOPSIS OF FIVE FATAL CASES OF YELLOW FEVER.

A brief account of the pathological appearances, both gross and microscopical, is given.

The disparity between the findings in the five cases recorded, and their deviation in certain respects from the classical description of yellow fever, are strikingly brought out in the tabular statement appended. It would, however, be somewhat premature, in the present state of our knowledge to set up a pathological standard of macroscopic and more particularly of microscopic appearances in West African yellow fever.

Macroscopic appearances—

Jaundice.—This is variable in tint, is most constant in the sclerae and is usually of a lemon-yellow colour.

Petechiae.—They are common in the facial area but may be diffuse.

Liver.—Pale yellow-brown in colour, with at times a greenish tinge.

The organ may be enlarged. The margins are firm. The cut surface has a dry dense appearance and the edges remain sharp and distinct. A hand lens is desirable for the examination of the cut surface. The lobulation can be made out in some cases, the central veins being apparently surrounded by pale areas giving place to reddish coloured zones peripherally. In the cases in which necrosis has occurred to

a more advanced degree, there is a uniform yellowish-brown appearance. Faint mottling may be present. The tissue is not especially friable and may even show a certain degree of elasticity. Fine subcapsular ecchymoses may occur.

Spleen.—Beyond congestion, this organ shows little change.

Kidneys.—The perinephric fat may show a bilious tinge. The capsule strips easily, leaving a smooth matt surface with well-marked stellate veins. In cut section the cortex appears to be increased and suggests an early degree of fatty degeneration. The amount of bile staining is usually small. Recent blood clots in the pelvic region were found in one case. The glomeruli frequently stand out sharply as minute congested points.

Stomach.—The contents are usually darkly stained and are soupy in consistence. Mucus shreds are visible. After washing, a fairly characteristic condition is seen, in the nature of haemorrhagic areas, varying in position and punctate in character. These haemorrhages tend to be situated on the crests of the mucous plicae, so that a linear or striped appearance results.

Duodenum.—May show an appearance similar to that seen in the stomach, but a more diffusely injected condition is more usual. The ampulla of Vater participates at times in the inflammatory congestion.

Small intestine.—Haemorrhages may be present in the wall.

Mesenteric glands.—They may be slightly enlarged and congested.

Heart.—The muscle tissue is somewhat flabby and lustreless. When opened into the muscle is dull and opaque-looking. There is no other evidence of degenerative change. The heart valves and the ascending aorta frequently show an icteric tint.

Lungs.—Oedematous and congested, especially posteriorly. Blotchy sub-pleural haemorrhages rather similar to those seen in purpura haemorrhagica are found mainly along the posterolateral and apical aspects. A frothy blood-stained fluid exudes from the cut surface. Slight crepitation is present. A lobar pneumonia was present in one case.

Post-mortem records—

Mrs. E. (1½ hours after death).

General.—Very corpulent. Skin greenish yellow in tint, also sclerae.

Cyanotic areas on forehead, abdomen and limbs.

Thorax.—No excess fluid in pleural or pericardial cavities. Chronic adhesions left pleura. Minute petechiae under parietal and visceral layers. Lungs markedly congested. When cut, a blood-stained frothy fluid exudes. Dark haemorrhagic areas scattered throughout the organs. These areas are not sharply defined from the surrounding tissue, nor do they project above the lung surface. Pieces of lung taken from different regions float in water. No bronchial inflammation. Trachea showed considerable inflammation.

Muscular tissue of heart soft and flabby. A few small petechiae on surface. When opened, the endocardium particularly in the region of the valve cusps distinctly jaundiced.

Abdomen.—No excess fluid, no petechiae.

The stomach showed blotchy congestive areas when opened. These occupied the lesser curvature and faded gradually into

the surrounding mucosa. No definite haemorrhages seen. Contents of organ watery, with mucus shreds and darkly stained flakes floating therein.

Duodenum, diffusely injected and of a dark red colour. Nothing of note in remaining portions of small intestine, except that in the caecal region there was slight congestion, with bile-stained contents.

Liver slightly enlarged with a smooth surface. In cut section the colour was light buff, uniform throughout with the exception of diffusely scattered minute red dots. These could not be identified with the central veins. The normal lobular formation was lacking.

The gall-bladder was partly filled with a thick viscid bile. Pancreas congested.

Spleen normal in size, dark in colour. In section the pulp was very dark, the lymph follicles showing up very clearly. Kidneys, slightly enlarged and soft in consistence. The surrounding fat was definitely jaundiced. Capsule thin and stripped easily. When sliced open, the cortex was relatively wide, swollen and of an opaque appearance. The cut margin of the organ tended to evert. The glomeruli could be discerned in places as minute congested points. A sharply-defined wedge-shaped area, dark in colour was present in the left kidney. A renal vein connected with this area was found thrombosed. The thrombus was a recent one.

Adrenals apparently normal.

Bladder showed hypertrophy of walls.

Mr. C. (1½ hours after death)—

General.—Well-built. No wasting. Icteric tint marked on face where petechiae also present. No rigor mortis.

Thorax.—No excess pleural fluid. Lungs voluminous and oedematous. Hypostatic congestion posteriorly, and also darkly coloured haemorrhagic areas scattered all through the organs. The tissue is crepitant and crackles when cut, with exudation of a frothy fluid. The lungs float in water. The oedema and haemorrhages are more marked in the right lung.

Heart: Muscle tissue appears firm, but when cut it presents a dull clouded appearance. No haemorrhages seen. Slight chronic endocarditis of mitral valve. Some early atheromatous patches in aorta.

Tonsils swollen but firm. Submucous haemorrhages present in trachea.

Abdomen.—No excess peritoneal fluid.

Stomach: Fluid contents tarry in colour, with darkly stained mucus shreds. Mucosa congested, with small ecchymotic areas, especially at the pyloric end.

Duodenum: Congestion in upper portion. No haemorrhages seen.

Liver: Normal size. Pale brownish-yellow in colour. Markedly firm and elastic. The cut surface resembles box wood and is dull and dry. The surface is quite uniform and no haemorrhages are visible. The normal liver structure cannot be made out.

The gall-bladder is collapsed.

Spleen: Normal in size, congested on cut surface, with prominent malpighian bodies.

Kidneys: Normal shape and size. Capsule strips easily. Cut surface is firm but cloudy looking. Cortex and medulla show a normal relationship.

Adrenals normal.

Bladder contracted, containing only an ounce or so of urine. No evidence of inflammation.

Mr. M. ($1\frac{1}{2}$ hours after death)—

General.—A lean small-framed man. Rigor mortis has not set in. The skin over the entire body is lemon yellow in colour, as are also the sclerae. A number of petechiae on chest and thighs. The cut surface of the skin is yellow in the dermis. There is almost no subcutaneous fat.

Thorax.—Pleural surfaces free from exudate. Right lung shows extensive consolidation in its lower, middle and a part of its upper lobe. Left lung shows a moderate amount of hypostatic congestion. Heart muscle firm. Its cut surface is pale brown. The valves have a yellow colour. The aorta is atheromatous.

Abdomen.—The sharp free edge of the liver extends three inches below the right costal margin. The organ is firm. The external surface shows a good deal of brown and red in the general yellow colour. The cut surface on being scraped is decidedly yellow. The lobules appear small and irregular in size and poorly defined. The bile in the gall-bladder is dark and stringy.

The kidneys are red. The capsules strip easily. Cortex and medulla can be distinguished. In the medulla, particularly of the left kidney there are a number of small haemorrhages.

Some scattered petechiae are seen in the cortex. Mucosa of pelvis decidedly yellow.

The spleen is somewhat enlarged. It is firm and the cut surface is blotchy.

The stomach contains about four ounces of very dark thick liquid.

The mucous membrane is slightly reddened and shows many minute redder spots.

The duodenum appeared normal.

The pancreas and the bladder showed nothing noteworthy.

Mr. A. ($6\frac{1}{2}$ hours after death)—

General.—Body well-developed and nutrition good. The skin over the entire body is light yellow in colour, except on the back where it is obscured by P.M. lividity. The sclerae are yellow. The cut surface of the skin is yellow but the dermis is white.

Thorax.—Dense pleural adhesions cover the entire left lung. The right lung is free. The cut surface of both lungs is frothy. Particularly in the right there are small irregular haemorrhagic patches with here and there small areas showing broncho-pneumonic changes. The heart muscle is firm. The epicardium shows a number of petechiae. The cut surface of the muscle is pale brown with just a trace of yellow. The valves are faintly icteric.

Abdomen.—The liver edges are sharp. The organ appears only slightly, if at all enlarged. The external surface is mottled, some areas being yellow and others purple. The organ cuts very easily. The cut surface oozes a red material which on scraping off leaves a yellow colour. The lobules can be distinguished showing three zones, a peripheral greyish-yellow, an intermediate red ring and a central portion of a greyish-yellow colour.

The kidneys are reddish-brown. The capsule strips with difficulty. The cut surface shows medulla and cortex clearly. Where cortex joins medulla there is a faint yellow zone. The medulla shows red streaks. The mucosa of the pelvis is icteric and there are a few scattered petechiae.

The stomach contained a dark liquid in which floated dark red flakes. The mucosa was a deep pink, most marked along the rugae.

The duodenum also showed a pink mucosa.

The spleen was enlarged. The cut surface was smooth.

The pancreas and the bladder showed no abnormality.

Miss U. (one hour after death)—

General.—Body well-preserved. Generalised dull greenish tint most marked on the sclerae. Small petechiae present, mostly on the face.

Thorax.—Lungs oedematous and congested, particularly posteriorly. Small subpleural haemorrhages on apical and postero-lateral aspects. Extensive fibrous adhesions on right side.

The heart is firm and its colour is pale. When opened the valves are seen slightly stained a yellowish-green. There was no evidence of myocardial degeneration.

Abdomen.—No excess fluid present. Discoloured patches discretely disposed throughout the small intestine.

The stomach contained about eight ounces of a black soupy fluid in which mucus shreds were floating. Punctate haemorrhages were present. In the region of the cardiac orifice these were grouped to form linear tracks, corresponding to the summits of the mucous folds. This linear formation was very striking.

The duodenum was diffusely congested. The ampulla of Vater was markedly congested and inflamed. Pressure on the gall-bladder caused a ready flow of normal-looking bile.

The liver was definitely enlarged. The colour was a pale straw-green. The consistence was firm. The cut edges remained sharp. With the aid of a lens, pale areas could be seen around each central vein, surrounded by a congested zone.

The spleen was soft and congested. The malpighian bodies were prominent.

The kidneys were large, their size increasing on removal of the capsule. Cortex large and pale. Recent haemorrhages were noted surrounding the ureter and vessels as they entered the organ at the hilum.

The adrenals appeared normal.

The pancreas showed nothing abnormal.

A HISTOLOGICAL ANALYSIS OF FIVE FATAL CASES OF YELLOW FEVER.

The organs showing the most marked changes are the liver, the heart and the kidneys. Even in these "key" organs, great variation of changes is presented.

Liver.—The amount of degeneration (fat staining) varies within wide limits. There may be only a peripheral fatty zone or the process may involve the whole lobule so that nothing is distinguishable but the portal tracts.

In one case there was a marked peripheral and also a central fatty zone. In between was a mid zone devoid of fat. At first sight this last appeared to be an unaffected area. Such was not the case, however, as an examination of haematoxylin-eosin-stained sections showed that this zone exceeded the others in regard to degenerative changes: it was composed of cell remnants, dilated capillaries and haemorrhagic areas (the mid-zonal necrosis of da Rocha Lima). This was fairly well shown in two cases.

Heart.—Three of the five cases showed complete fatty degeneration of the muscle fibres. The other two were entirely negative. In all, vascular congestion was present.

Kidneys.—Fatty degeneration was well-marked in two cases. In these the tubular condition suggested a sub-acute inflammation, in that the cells lining the tubules were "low" in character, the lumina of the tubules being correspondingly wide. In such cases the tubular contents consisted mainly of hyaline globules of varying size, massed together in the collecting tubules to form casts. In some of the casts a fairly definite structure was visible (*i.e.*, cellular casts).

The glomeruli all showed congestive changes, accompanied as a rule by fibrinous exudation into the glomerular space, the lining membrane of which showed hypertrophic changes. Lime casts were not noted. The vessels shared in the congestive changes but only in one case were definite haemorrhages present.

Spleen.—The interpretation of pathological changes in this organ is always difficult, and this difficulty was increased in some of the present cases owing to the presence of malarial pigment. Haematoidin crystals were noted in one case. In two there was cellular exudation around the larger veins as they traversed the trabeculae.

Stomach.—In all the cases congestion of the vessels in the mucous and submucous layers was evident. Extravasations into the mucous layer were frequent. In places, superficial membranous patches were present, composed mainly of extravasated blood cells and necrotic mucous membrane.

The duodenum was involved in a much milder degree. The remainder of the alimentary tract showed nothing significant.

Lungs.—Haemorrhagic areas almost like infarcts were a constant finding. Apart from these intra-alveolar haemorrhages, there were frequently subpleural petechial extravasations. In one case a lobar pneumonia (grey hepatisation stage) was present on the right side. Hypostatic congestion was usually marked.

Nothing of interest was noted in other organs.

B. HÆMATOXYLIN EOSIN STAIN.

	Mrs. E.	Mr. C.	Mr. A.	Mr. M.	Miss U.
LIVER.	Parenchyma appears granular and vacuolated. Isolated tags of liver cells around portal tracts. Congestion of vessels in the tracts and a diffuse round cell infiltration also present. Nuclei in many cells are lacking, others show chromatolytic and pyknotic changes. When present they are mostly central. Capillaries full of blood cells are seen separating the adjacent rows of cells. Some lymphocytes have migrated through the parenchyma. Greenish-brown fine granules of pigment fill some of the hepatic cells and mask their structure.	From without there is first a layer of intact cells around most of the portal tracts. These give place to degenerated and finally to disintegrated cells. As the central vein is neared, a second fairly intact layer is seen. The part showing most change is mid-lobular. Fine greenish-brown stippling is seen in some cells. Dark green granules occur in many of the inter-cellular spaces.	Liver cells recognisable only around the portal tracts where they occur as a thin broken band. Centrally the cells become granular and vacuolated, with no nucleation. The intervening capillaries are greatly distended and have formed small scattered hæmorrhages. Considerable chronic infiltration (small round cells) is present around the portal tracts.	Normal lobulation is present. Some of the central veins are the site of small hæmorrhages but many are surrounded by a band of fairly well preserved liver cells. A similar band surrounds the portal tracts so that, sandwiched between the two layers is a zone of necrotic hepatic cells. The endothelial, Kupfer and some liver cells show well-marked greenish pigment granules. The portal tracts are infiltrated with lymphocytes, and the bile ducts contain cast-like structures, (inspissated bile?).	Portal tracts are surrounded by broken-up groups of intact liver cells mixed with cells showing various stages of degeneration. Round the central vein, liver cells are vacuolated and their nuclei are degenerated. Between the two zones is an area, varying in width, of necrotic liver cells, their outlines faint, their protoplasm vacuolated and the nuclei lacking. Lying in between these cell-ghosts and compressing them are the distended blood capillaries, sharply marked off by the swollen endothelial cells, from the necrotic groundwork. The portal tracts show a round cell infiltration. Pigmentary deposits are very scanty.
HEART	The muscle fibres are widely separated and the fibrous tissue between is oedematous. Vessels congested. Nuclei of cells degenerated and in many cases absent. The fibres have a clouded, granular appearance, their normal striation being lost.	Fibres separated and vessels congested. Most of the fibres show nucleation and brown pigment. Striation indistinct and fibres show granulation.	Nucleation present but the protoplasm of the muscle fibres is granular and devoid of striation. The vessels are greatly engorged and contain greenish-brown specks.	The fibres are distinctly granular but the normal striation can be distinguished. The vessels are congested.	Fibres cloudy in appearance, but the striation is visible. The nuclei stain faintly and show chromatolytic changes. Congestion of the vessels is present.
SPLEEN.	Marked vascular congestion and distension of sinuses. Halos of inflammatory cells (mainly lymphocytic) surrounding the veins in the trabeculae. Malpighian bodies enlarged and outline irregular. Two varieties of pigment, black granules sometimes obscuring the nuclei, and crystalline globules, probably hæmatoidin in the endothelial cells.	Congestion and sinusoidal distension. Malpighian bodies well-marked. Pigment laden cells numerous. Capsule thickened.	Marked congestion of splenic pulp. Pigment granules scattered throughout the organ.	Congestion marked. Generalised pigmentation.	Congestion of the pulp tissue with numerous pigmented cells. The pigment is in the form of irregular brown granules. The veins as they lie in the trabeculae show a slight amount of surrounding cell-exudation (mostly lymphocytes).
LUNGS.	Areas of hypostatic congestion. Hæmorrhages in scattered areas, filling the alveoli. There are areas of compensatory emphysema.	Hypostatic congestion and hæmorrhages present.	Capillary congestion and extravasations which fill the alveoli. Some of the areas resemble recent infarcts.	Consolidated area shows an early stage of grey hepatisation, the alveolar spaces being filled with polymorphs.	There are areas in which the alveoli are full of blood cells. These are surrounded by zones of compensatory emphysema. Sub-pleural hæmorrhages are present.
STOMACH.	Some hæmorrhagic extravasations into submucosa. Cells are swollen and many have disintegrated.	Extravasation into submucosa. Round-cell infiltration into loose connective tissue of villi. Epithelium lacking in places.	Vascular congestion of mucous and submucous coats. Some patchy necrosis of the epithelial cells.	Mucous membrane is congested, with superficial collections of red cells lying on it. Small hæmorrhages in mucosa.	Surface of mucous membrane is necrotic, and in many places small extravasations of red blood cells have occurred forming delicate membranous patches. Vessels of submucous and muscular layers are congested.
KIDNEY.	Tubular epithelium is as in acute nephritis. The epithelium is "high" its free surface is ragged, and by crumbling has formed a granular material which in the collecting tubules appears as homogeneous hyaline casts. Nucleation is fairly good, especially in the collecting tubules. There is marked congestion of glomerular tufts, and hypertrophy of the endothelium. Some glomeruli appear to be anchored to their capsule by a delicate fibrinous exudate. Vessels congested and contain fine greenish brown pigment. A similar but coarser pigment is free and also in many of the tubular epithelial cells. Capsule apparently normal.	Tubular epithelium "low", leaving wide lumina filled with an amorphous substance. Many nuclei are shrunken and distorted but some are in fair preservation. The tubular contents are mostly cellular. Dark green pigment granules occur. Glomerular capillaries congested, with much swelling of the endothelium and exudation into the glomerular space. Vessels congested and contain fine pigment. Capsule normal.	Tubular appearance is as that seen in cases of commencing sub-acute nephritis. Epithelial cells tho' high in type, do not completely fill up the lumina, which contain a granular material and hyaline casts. Most of the cells retain their nuclei; but their protoplasm is granular and vacuolated. The glomeruli are very congested, with slight exudate into the glomerular spaces. Cellular lining of Bowman's capsule is very prominent. Vessels congested. A few small extravasations. Capsule thin.	Tubular epithelium is "low" and lumina are distinct. They contain a scanty coarsely globular detritus. Some of the nuclei show chromatolytic changes. In some of the collecting tubules, homogeneously-stained casts appear. Capillary congestion is present, with some granular exudate into the glomerular spaces. The vessels are congested. Capsule shows slight irregular thickening.	Tubular epithelium is "low" in type, the lumina are relatively wide and contain irregular sized globules of a pink-staining substance. In the collecting tubules, these globules apparently coalesce to form the so called colloid casts. Epithelial cells are granular. Glomeruli congested. In many of the glomerular spaces, globules similar to those in the tubules are present. The lining epithelium is swollen, individual cells showing up here and there like miniature frog-spawn. A granular substance is also present in many of the spaces. Vessels tho' considerably congested, have not given rise to hæmorrhages. Capsule shows some oedema and vascular congestion.

HISTOLOGICAL CHANGES IN THE ORGANS OF THE INDIVIDUAL CASES.

A. FAT STAIN (SUDAN III).

	Mrs. E.	Mr. C.	Mr. A.	Mr. M.	Miss. U.
LIVER ...	Has undergone complete fatty change. The only parts not involved are the portal tracts. The lobular arrangement has completely disappeared. The intracellular fat globules show much variation in size. Around Glisson's capsule they are mostly small and give a stippled appearance to the cells but groups of larger globules occur. Here and there the larger globules are more numerous at some distance from the portal tracts. The fat-laden rows of cells are separated from each other by distended capillaries.	Presents a much milder parenchymatous degeneration than Mrs. E. The lobular arrangement is intact. The fatty degeneration appears as a narrow peripheral band in each lobule. The fat drops are very fine. Around each central vein is a zone of intact liver cells. As these are traced peripherally, they become more difficult to interpret and show patches of fatty stippling. Finally they merge into the peripheral band already referred to.	Shows well-marked diffuse fatty degeneration. The globules are fairly large and arranged as a rosette in many of the cells, around the centrally-lying nucleus.	There is diffuse fatty degeneration more definite at the periphery of the lobules. Around some of the central veins is an area which is devoid of fat. There is wide variation in the size of the fat globules. They tend to become smaller as the central vein is approached.	Presents a somewhat distinctive picture. There are two fatty zones. One is wide and well-marked, situated peripherally. The other, less distinct and narrow, clothes the central vein.
HEART ...	An exquisite example of myocardial degeneration. Every muscle fibre is crammed with fine fat droplets.	No fat found. Yellowish staining granules are seen at the nuclear poles.	Well-marked diffuse fatty degeneration. The muscle fibres are replete with fine fat droplets.	With the exception of yellow staining granules around the nuclear poles, no fatty granules are seen.	All the muscle fibres contain small fatty globules, <i>i.e.</i> a diffuse degenerative process.
KIDNEY ...	The bases of the cells are occupied by fatty globules of varying size. The endothelium of the glomerular tufts also shows slight fatty changes.	Faint traces of fat in the tubular epithelium.	Shows isolated tubules which have undergone fatty degeneration. No generalised fat change visible.	Very slight amount of fatty degeneration present.	The fatty globules are rather more numerous towards the bases of the tubular epithelial cells.

A NOTE ON THE CONDITION CALLED "BLACK TONGUE."

Dr. J. R. C. Stephens, Medical Officer, Ilorin, reported that there was a complaint prevalent among the natives of his district, called by themselves "black tongue," and that he thought it required investigation. Dr. E. C. Smith proceeded there on 1st October, 1926 and immediately began his investigations in the surrounding villages.

In all, twelve patients suffering from the condition were seen,

Six cases at Ifaki.

Three cases at Usi.

Two cases at Awtun.

One case at Ikun.

The cases at Awtun were watched from the day of development. At the onset, the tongue is found to be thickly coated with a white membranous-like layer, usually dry, but in some cases it may become slimy. Fragments of this membrane can be readily lifted away, leaving a smooth erythematous but non-haemorrhagic surface. The condition, apparently, does not cause any soreness in the tongue, but may bring about some slight distress in breathing, especially if it spreads to the soft palate.

The patients complain that it interferes with their sense of taste. They show their tongue to their friends who are usually found to be responsible for the diagnosis.

On or about the third day, the membranous formation commences to dry up and become discoloured, finally assuming a deep brown or black hue. This appearance is more particularly marked on the dorsum posteriorly, and on the sides of the organ.

At this stage the membrane may be very hard and dry, being separated with difficulty.

From the fifth day onwards, the condition shows signs of clearing up, the membrane breaking off and coming away in fragments.

It was noted that the formation of the membrane was accompanied by fever (100 to 103° F.) and by the presence of relapsing fever spirochaetes, in the patient's blood.

Scrapings from the affected tongues, made at various stages, revealed the presence of a large branching yeast-like fungus, probably of the "oidium" type. It was obtained in pure culture from four cases.

Conclusions—

- (1) Apparently the condition known among the natives as "black tongue" or, in the early stages, "white tongue," is due to the active proliferation of a fungus, probably of the oidium type, coincident with fever and the presence of the spirochaetes of relapsing fever in the patient's blood.
- (2) The lingual condition may persist during the apyretic periods between the relapses, that is at a time when no spirochaetes will be found in the blood and the temperature will be normal.
- (3) It would seem that the condition is worth bearing in mind as a possible indication of the presence of relapsing fever in a district.
- (4) It is the natives themselves who complain of the condition and who have given it its descriptive name.
- (5) It is possible that the condition may arise in other febrile diseases or in periods of ill-health, although no such evidence was obtained in the short space of time available.

LEPROSY.

The treatment of the inmates of the Yaba Leper Asylum was continued until the first week in October when they were handed over to the Medical Officer appointed to take charge.

Ethyl hydriocarpate which had been in use from October, 1925, was discontinued at the end of January. There was no obvious superiority in its action over that of moogrol and as the patients themselves preferred the latter drug, its intra-muscular administration was recommenced.

Thirteen patients received treatment at weekly intervals. All, with the exception of two cases, were old-standing, well-advanced cases that had been under treatment for years.

The following are the details:—

David—(mentioned in Reports of 1916, 1917, 1918, 1920, 1921 and 1924). Had twenty-four doses at weekly intervals, with a total of twelve weeks' rest. No active signs, nodules and thickenings have disappeared.

Lawani—(mentioned in Reports of 1916, 1917, 1918, 1920, 1921 and 1924). Tubercles fewer and less prominent, ulcerations tending to heal after eighteen doses at weekly intervals with eighteen periods of one week's rest.

Calabar—(mentioned in Reports of 1917, 1918, 1919, 1920, 1921, 1922 and 1924). Had twenty-four weekly doses with twelve periods of rest. The tubercles are less prominent, and broken skin has healed.

Brimah—(mentioned in Report of 1924). Has had twenty weekly doses with sixteen periods of rest. The acute progress of the disease has been arrested.

Idowu—(mentioned in Report of 1924). Has had twenty-two weekly doses with fourteen periods of rest. In this case, also, the acute process has been arrested.

Ogunbiye—(mentioned in Report of 1924). Has submitted to only nine doses. Active signs of the disease are in abeyance.

Oyekan—(mentioned in Report of 1924). Has had fourteen doses. An ulcer on the leg is the only outward sign of active disease.

Aina—(mentioned in Report of 1924). Has had sixteen doses. Condition is apparently stationary.

Patience—(mentioned in Report of 1924). Had only nine doses. Died of intercurrent disease in September.

Mowunmi.—A female aged about fifty-five years, admitted in the latter end of 1925, destitute, with signs of nerve leprosy, absorption of the bones of fingers and toes with " nibbling " of their extremities, and several maculae. Was given fourteen doses moogrol with twenty-two periods of rest. Absorption does not appear to have progressed, and the tips of the fingers and toes are cleaner.

Salami.—An old inhabitant of the Asylum who had persistently refused all treatment. He allowed himself to be persuaded in August and he received five doses.

Ojo.—An early case, had twenty-two doses with fourteen rests. The slight tuberculation on the face has distinctly lessened, and the tubercles on the ears have practically disappeared.

Abia.—Also an early case in a young male adult as in Ojo (above). There is a slight tuberculation on the cheeks and on the tips of the ears. The skin generally has the appearance of urticaria. He began treatment on 13th September, 1926, and has received three doses.

The experience of moogrol during the year has demonstrated again the suitability of that drug in the treatment of leprosy at Yaba. It is well tolerated by all and if taken over a sufficiently long period, and with regularity (once a week), it has a very definite remedial effect. Different individuals react differently, and a suitable dosage has to be found for each patient.

ENTOMOLOGY.

The work in this section was done by Mrs. Summers Connal.

Particular attention was devoted to the markings of the common household mosquito, *Aedes argenteus* which used to be better known under its old title of *Stegomyia fasciata*.

Great variation was found to exist and the findings have been summarised for this report.

Specimens of biting insects were gratefully received from the following:—

Dr. Allan, Major Bell, Dr. Crawford, Dr. Dyce-Sharp, Dr. Fraser, Dr. Orpen, Dr. Pasqual, Mr. Pretious, Dr. Smith, Dr. Stephens, Mr. Till, Dr. Waller and Mr. G. Wilson.

ON THE NUMEROUS VARIATIONS OCCURRING IN THE SPECIMENS OF *Aedes argenteus*, POIRET OBTAINED IN LAGOS, NIGERIA.

During the last few years there has been a considerable increase in the number of new varieties of mosquitos created. This has made the path difficult for the Entomologist working abroad and has rendered things perplexing to Medical Officers in general.

On reading over again the description of *Stegomyia fasciata* var. *atrifasciata* Edwards ⁽¹⁾ it appeared that it would be interesting to examine a number of freshly hatched and therefore undamaged specimens of *Aedes argenteus*, and to note how many conformed to the original description.

No difficulty was experienced in obtaining the necessary material, as collections of larvae are received daily from the Medical Officer of Health in Lagos.

One thousand males and one thousand females of the species were mounted in the order of their emergence from the pupal case, and were examined under the binocular microscope.

The one really constant feature was found to be the lyre on the thorax, although even there the outline and the centre lines varied in breadth and in colour, and the centre lines also varied in length.

But as the abdomen and the hind legs showed the greatest variations, the present paper is restricted to a description of these.

There were specimens with the abdomen all black, except for the lateral spots. Again there were many which had the abdomen covered with pure white scales. In others, white or yellow scales were scattered all over the abdomen so as to give a brindled appearance. Some examples had both apical and basal bands and others had only basal bands.

The depth of the bands varied within wide limits. Many of the basally-banded had a narrow band of perhaps two or three rows of scales on the second and third segments which increased to five rows on the fourth and fifth segments. Some had deep bands on every segment, others had narrow ones of only one row of scales.

The first segment of the abdomen was all black in some cases, and all white or all yellow in others.

In some individuals there was a triangular median spot on the first abdominal segment, the colour of which was white or yellow. The lateral spots varied in size and occasionally they were purple in colour.

The seventh segment usually had one row of yellow apical scales, but sometimes this whole segment was covered with white or yellow scales, and in a number of instances the coloured scales formed a triangular patch. There was variation in colour both of the groundwork and of the ornamentation. Both white bands and yellow bands were met with. The groundwork was not always black, and light brown shades occurred.

As regards the hind tarsus, there were numerous variations. In a large number the last segment was typically all white. In others the tip was black, in others again the terminal half or the terminal quarter was black. Several specimens thought to be the variety *atritarsis* of Edwards had the typical white band on the last segment reduced to a mere spot. The white band on the fourth segment of the hind tarsus varied in breadth not only in relation to its own segment but also in relation to the bands on the other segments. In fact the length of the fourth segment itself varied, for in some cases it was extremely short, being just a little longer than the fifth segment whilst in other specimens it was almost twice as long. The breadth of the white band on the fourth segment varied between two extremes, on the one hand leaving only the tip black and on the other being reduced to a white basal spot. In some instances the white band was a quarter, in others a half and in some three-quarters of the length of the fourth segment. It was often difficult to say whether or not the segment was all white, not only in those cases in which it was short but also in the cases in which it was long, in relation to the other segments.

The breadth of the band on segments one, two, three and four varied in relation to each other. Typically, according to description, the single bands on each of the first four segments of the hind tarsus are equal in breadth. Two variations, however, excluding variety *atritarsis*, occur quite commonly. In one, the breadth of the band on the fourth segment is double that of each of the first, second and third segments. In the other, the band on segment four equals that on segment one, but each is double the width of the band on segment two and segment three.

The colour of these bands was almost constantly white, but in one or two cases it was yellow.

The groundwork of the hind legs was often brown instead of black.

Female Specimens.—In the thousand females there were 689 (*i.e.*, 68.9 *per cent.*) with typically basally banded abdominal segments. Of these, 390 had the last segment of the hind tarsus typically white. The remaining 299 had the last segment of the hind tarsus with a black tip, or the terminal quarter, or terminal half, black, and in some almost the whole segment (variety *atritarsis*) was black. Therefore, the percentage of typically basally banded females, equalling 68.9 *per cent.*, showed typical markings on the last segment of the hind tarsus in 56.45 *per cent.*

Specimens with a "brindled" abdomen came next in numerical order. These had usually apical as well as basal bands with a broad median longitudinal band uniting them, the remainder of each segment being dotted with white or yellow scales. The "brindled" numbered 127 and in eighty-one the last segment of the hind tarsus was white. In forty-six the last segment of the hind tarsus showed the variations described in the first group.

Individuals with both apical and basal bands were 110 in number. These showed no white or yellow scales elsewhere on the abdominal segments. In sixty-seven the last segment of the hind tarsus was all white and in the remaining forty-three it was not all white. The width of the abdominal bands in this third group varied greatly. In some the apical band consisted of only one row, in others there were three or four rows in the band. In some instances, both the apical and the basal bands were so broad that only a narrow black strip was left between them. It was seldom that the basal and apical bands were equal in breadth.

The most interesting and beautiful group consisted of those which had the abdomen covered with white scales. Occasionally a specimen showed a few black scales amongst the white, and including these there were forty in the "white abdomen" group. Of these, twenty-six had the last hind tarsus segment all white and in fourteen it was not white. There were no examples of variety atritarsis in this group but in several the terminal half, and in others the terminal quarter, was black.

The fifth group contains the specimens which had the abdomen all black. There was no question of the bands having been rubbed off. Each specimen was perfect and intact. The scales were all black or purplish black. With the light at a certain angle it was sometimes possible to trace a purple basal band on the black background. The lateral spots on these specimens were usually white, but in one or two cases they were purplish black. They numbered twenty-five, and seventeen had the last hind tarsal segment white. In the remaining eight some had the last segment half black, but there was no example of variety atritarsis amongst them.

The smallest and last group was made up of nine individuals with only apical bands on the abdominal segments. The banding was narrow, with usually only one row of white or yellow scales in each. Two of these had the last hind tarsal segment white. The other seven nearly all had the terminal half black.

Table VII condenses the information.

TABLE VII.

Basally banded	...	...	689	Last segment, hind tarsus white	390.
Brindled	...	...	127	"	81.
Apically & Basally banded	...	...	110	"	67.
White abdomen	...	...	40	"	26.
Black abdomen	...	...	25	"	17.
Apically banded	...	...	9	"	2.
1,000				583.	

Male Specimens.—The largest group consists, as it does in the females, of those with a basally banded abdomen. They numbered 852, a much larger proportion than was found in the females. Of this number, 477 had the last hind tarsal segment white.

The second largest group is formed by the apically and basally banded abdomen, thus differing from the females, whose second group consisted of the "brindled" abdomen. There were seventy-four in this second group, and of these there were forty-nine which had the last segment of the hind tarsus white.

The third group comprises the "brindled" specimens, of which there were forty-six and amongst these there were twenty-five with a white last hind tarsal segment.

Specimens with a white abdomen form, as they do in the case of the females, the fourth group. They numbered only twenty-one and they included fifteen with the last hind tarsal segment white.

The black abdomen group was made up of seven individuals, four of which had the last segment of the hind tarsus white.

The fifth group, the apically banded was unrepresented in the males. Table VIII gives the figures.

TABLE VIII.

Basally banded	852.	Last segment hind tarsus white	477.
Apically and basally banded	74.	" " " "	49.
Brindled	46.	" " " "	25.
White abdomen	21.	" " " "	15.
Black abdomen	7.	" " " "	4.
	<u>1,000</u>		<u>570</u>

It will be seen that in the females the typically marked abdomen was found in 68.9 *per cent.* The typical last hind tarsal segment occurred in 58.3 *per cent.* A typical abdomen and last hind tarsal segment, however, was present in only thirty-nine *per cent.*

The following figures show the frequency of typical markings on the last hind tarsal segment in the several atypically ornamented abdomens:—

Basally banded	56.60 <i>per cent.</i>
Brindled	63.70 "
Apically and basally banded ...	69.90 "
White abdomen	65 "
Black abdomen	68 "
Apically banded	56.60 "

As regards the males, 85.2 *per cent.* showed a typically marked abdomen, and fifty-seven *per cent.* showed a typical last hind tarsal segment. A typical abdomen and last hind tarsal segment were found in 47.7 *per cent.*

The percentage of typical marking on the last hind tarsal segment in the different groups of atypically marked abdomen is shown below:—

Basally banded	55.98 <i>per cent.</i>
Apically and basally banded ...	66.21 "
Brindled	54.34 "
White abdomen	71.42 "
Black abdomen	57.01 "

Markings on the hind tarsus.—There were three distinct groups in the markings on the hind tarsus. (The last hind tarsal segment was considered separately). The most frequent markings were the band on the second and third segments equal in breadth, and the band on the first and fourth segments also equal but each of them double the breadth of that on the second and third segments. In this group there were 519 females and 529 males. (Group 1).

In the second group, that in which the bands were all of the same breadth, on the four segments, there were 236 females and 417 males. (Group 2.)

In the third group, the bands of the first, second and third segments were all equal but that on the fourth segment was double the breadth of each. In 238 females and forty-seven males this arrangement was present. (Group 3.) The markings of variety atritarsis were noted in seven females and seven males.

BREEDING EXPERIMENTS.

Several breeding experiments were undertaken, the details of which are given below.

Experiment (1)—Female with “ white ” abdomen.

Male with narrow basal bands.

The last hind tarsal segment in both was all white.

The banding of the first, second, third and fourth segments of the hind tarsus conformed to Group 1 in the female, and Group 2 in the male.

Sixteen adults were obtained: ten females and six males.

In the females—

The abdomen was basally banded in five.

The abdomen was brindled in three.

The abdomen was apically banded in one.

The abdomen was white in one.

In the males—

The abdomen was basally banded in two.

The abdomen was brindled in two.

The abdomen was apically banded in one.

The abdomen was white in one.

Thus the abdominal markings of the father were reproduced in seven of the progeny, those of the mother in two, and seven showed the marking of neither parent.

The last hind tarsal segment had a black tip in 3♂, 1♀, was all white in 3♂, 8♀ and was half black in one female.

In the females the hind tarsal markings belonged to Group 1 in seven cases, to Group 2 in three cases.

In the males the hind tarsal markings belonged to Group 1 in two cases, to Group 2 in four cases.

They were thus rather evenly divided, nine favouring the mother and seven the father.

Experiment (2)—Female with white abdomen.

Male apically and basally banded.

Last segment hind tarsus, female all white.

Last segment hind tarsus, male with black tip.

The banding on the hind tarsal segments in both belonged to Group 2.

Six adults hatched out, five males and one female.

In the males—

The abdomen was apically and basally banded in three.

The abdomen was white in one.

The abdomen was brindled in one.

In the single female the abdomen was apically and basally banded.

In the males, three had the last hind tarsal segment all white and two had a black tip. In the female also there was a black tip.

In the males the hind tarsal segments fell into Group 2 in three cases, and into Group 1 in two cases.

In the female the bands were all equal (Group 2).

Experiment (3)—Female with white abdomen.

Male with broad white basal bands.

In the female, the last hind tarsal segment was black-tipped.

In the male, the terminal half was black.

In the female, the hind tarsal segments fell into Group 2.

In the male they came into Group 1.

Thirteen adults hatched out, eight males and five females.

In the males—

The abdomen was apically and basally banded in four.

The abdomen was basally banded in two.

The abdomen was brindled in one. And in the last there were a few white apical scales on each segment, in addition to the basal banding. The basally banded specimens had the bands very narrow. All the bands were white. In the females, all had both apical and basal bands but in three, the colour of the bands was yellow, not white.

In the males the last hind tarsal segment was black-tipped in six, was all white in one, and in the last, the terminal half was black.

In the females there was a black tip in three and the terminal quarter in two cases was black.

In the males the hind tarsal segments fell into Group 2 in seven cases, and into Group 1 in one case.

In the females all came into Group 2.

Experiment (4)—Female with white abdomen.

Male brindled.

In the female, terminal half, last hind tarsal segment black.

In the male, merely the tip was black.

In the female, the hind tarsal segments came into Group 1.

In the male, they came into Group 2.

Two adults hatched out, both females. One had broad basal bands, and the other had a white abdomen. The last hind tarsal segment in both was black-tipped. The hind tarsal segments fell into Group 2 in both cases.

Experiment (5)—The parents in this experiment were both hatched out from Experiment (1).

The female had narrow basal bands on the abdomen.

The male abdomen was white.

The last hind tarsal segment in the female was all white.

In the male it had a black tip.

The hind tarsal segments in both parents came under Group 1.

Seven adults were obtained, four males and three females.

In the males—

The abdomen was apically and basally banded in three.

The abdomen was white in one.

In the females—

The abdomen had narrow basal bands in two.

The abdomen was brindled in one.

The last hind tarsal segment, in the males, had the terminal half black in two cases, in one the tip was black and in the other the segment was all white.

In the females, all three had the last hind tarsal segment all white. The banding of the hind tarsal segments in the males, came into Group 2 in three cases and in the fourth it was in Group 1.

In the females, two came into Group 2 and one into Group 1.

Experiment (6)—Here also, the parents hatched out in Experiment (1).

Both parents had narrow basal bands on the abdomen.

Both also had the last segment of the hind tarsus white.

In the female, the banding of the hind tarsus came into Group 2. In the male, it came into Group 1.

Three adults hatched out, two males and one female. All had narrow basal abdominal bands. One male had the last hind tarsal segment with the terminal half black and the other had only the tip black.

In the female, the last hind tarsal segment was all white.

In the males the markings on the hind tarsus came under Group 1, and in the female under Group 2.

Experiment (7)—The parents again came from Experiment (1).

Both had a brindled abdomen.

The female had the last hind tarsal segment all white.

The male had the tip black.

The hind tarsus of the female came into Group 1.

That of the male came into Group 2.

Only one adult was obtained, a female. The abdomen was basally banded, the bands being narrow. The tip of the last hind tarsal segment was black, and the banding of the hind tarsus brought it into Group 2.

Several attempts were made to get adults of the next generation, but the females invariably died before eggs were laid.

Although it seemed fairly certain that light and shade, and different colours of the breeding-dishes were not the reason of the variations in the markings, several experiments were carried out with this possibility in view.

A pure white, an unrelieved black and a reddish brown environment were tried.

The white breeding-dishes yielded no more insects with abundant white ornamentation than did the black, nor did the black dishes contain more darkly marked specimens than the white. The reddish brown dishes had no obvious influence on the markings.

SUMMARY AND CONCLUSIONS.

The markings of *Aedes argenteus*, Poiret, are dealt with.

Attention is directed to the abdominal markings and to the markings on the last hind tarsus.

One thousand males and one thousand females of the species were examined.

There was no selection, the insects being taken as they hatched from the pupa in collections sent daily by the Medical Officer of Health, Lagos. As regards the abdomen, the following groups were encountered, among the females:—

- (1) Basally banded.
- (2) Brindled.
- (3) Apically and basally banded.
- (4) All white.
- (5) All black.
- (6) Apically banded.

In the males no specimen of the apically banded was met with.

These variations were found to be unconnected with the colour of the breeding dish, in so far as black, white and reddish-brown surroundings are concerned.

Nor were the markings found to vary with age or feeding. The same markings were found on insects newly hatched as on those kept alive for a day or two and given a meal on blood or on fruit juice.

The last hind tarsal segment may have a black tip, or the terminal quarter or half may be black, or as in variety atritarsis there may be a mere white basal spot.

The white bands on the first four segments of the hind tarsus fall into three groups:—

- (1) The bands are equal in breadth.
- (2) The band on the fourth segment is double the breadth of each of those on the other segments.
- (3) The band on the fourth segment is equal in breadth to that on the first, and each is double the breadth of those on the second and third segments.

There appeared to be no justification for describing a new variety from any of the specimens which were examined.

The findings recorded above are given in the hope that they will tend to discourage the creation of new varieties.

(1) Reference, Edwards, F. W. Bull. Ent. Res. Vol. X, 1919-1920, page 129.

MOSQUITO LARVAE FROM THE LAGOS MUNICIPAL AREA.

Owing to the absence of the Honorary Entomologist, no identifications of mosquito larvae were recorded in the period March to July or in parts of February and August.

There were 1,389 collections of larvae sent for identification, from twenty-six different receptacles or sources, and containing nine different species, in the period January-February, and September-December.

The species were:—

<i>Aedes argenteus</i>	...	in 697 collections.
<i>Culex nebulosa</i>	...	„ 634 „
<i>Culex fatigans</i> ...	...	„ 58 „
<i>Anopheles gambiae</i>	...	„ 14 „
<i>Culex decens</i> ...	...	„ 7 „
<i>Culex thalassius</i>	...	„ 5 „
<i>Aedes luteocephalus</i>	...	„ 4 „
<i>Culex duttoni</i> ...	...	„ 3 „
<i>Eretinopodites chrysogaster</i>	„	1 collection.

Two or more species occurred in the same collection as follows:—

Barrel.	<i>Aedes argenteus</i> and <i>Culex fatigans</i>	...	...	in 1 collection.
Canoe.	<i>Culex nebulosa</i> and <i>Culex thalassius</i>	...	...	„ 1 „
Catchpit.	<i>A. argenteus</i> and <i>C. nebulosa</i>	...	...	„ 2 collections.
	<i>C. fatigans</i> and <i>C. nebulosa</i>	...	...	„ 1 collection.
Cooler.	<i>C. nebulosa</i> , <i>A. argenteus</i> and <i>C. fatigans</i>	...	...	„ 1 „
Drum.	<i>A. argenteus</i> and <i>C. nebulosa</i>	...	...	„ 1 „
Mortar.	„ „ „ „ „ „	...	...	„ 1 „
Pool.	<i>C. fatigans</i> and <i>Anopheles gambiae</i>	„	...	„ 1 „
Pot.	<i>A. argenteus</i> and <i>C. nebulosa</i>	...	...	„ 9 collections.
	„ „ „ „ <i>fatigans</i>	...	...	„ 4 „
	„ „ „ „ <i>A. luteocephalus</i>	...	...	„ 2 „
	<i>C. nebulosa</i> and <i>C. fatigans</i>	...	...	„ 2 „
	<i>A. gambiae</i> and „ „	...	...	„ 1 collection.
	<i>C. duttoni</i> and <i>C. decens</i>	...	...	„ 1 „
Tin.	<i>A. argenteus</i> and <i>C. nebulosa</i>	...	...	„ 1 „
	„ „ „ „ <i>A. luteocephalus</i>	...	...	„ 1 „
	<i>C. fatigans</i> and <i>C. decens</i>	...	...	„ 1 „
	<i>Eretinopodites chrysogaster</i> , <i>C. fatigans</i> and			
	<i>A. argenteus</i>	...	...	„ 1 „
Tyre	<i>A. argenteus</i> and <i>C. nebulosa</i>	...	...	„ 1 „
	<i>C. fatigans</i> „ „ „	...	...	„ 1 „

The combinations of two larvae are :—

Aedes argenteus and Culex nebulosa	...	...	...	in 15 cases.
" " " " fatigans	...	...	...	" 5 "
" " " Aedes luteocephalus	...	...	...	" 3 "
Culex nebulosa " Culex fatigans	...	...	...	" 4 "
Anopheles gambiae and Culex fatigans	...	...	...	" 2 "
Culex decens and Culex fatigans	...	...	...	" 1 case.
" " " " duttoni	...	...	...	" 1 "
" nebulosa and Culex thalassius	...	...	...	" 1 "

The combinations of three larvae are :—

Culex nebulosa, Culex fatigans and Aedes argenteus	...	in 1 case.
Eretinopodites chrysogaster, C. fatigans and A. argenteus	" 1 "	

The sources or receptacles were twenty-six in number, namely :—

Barrel, bottle, bucket, calabash, canoe, catch-pit, cooler, drain, drum, dye-pot, flower-pot, grinding stone, jug, kettle, mortar, pail, pan, pit, pool, pot, tannery pit, tank, tin, tree-hole, tyre and well.

The sources or receptacles in their order of attraction were :—

Pot	...	...	855 collections.
Catchpit	...	...	134 "
Tin	...	...	102 "
Drum	...	...	57 "
Barrel	...	...	44 "
Bucket	...	...	28 "
Drain	...	...	25 "
Bottle	...	...	21 "
Well	...	...	19 "
Pool	...	...	17 "
Jug	...	...	13 "
Kettle	...	...	10 "
Pan	...	...	9 "
Tyre	...	...	9 "
Tank	...	...	8 "
Cooler	...	...	7 "
Canoe	...	...	7 "
Pail	...	...	6 "
Mortar	...	...	4 "
Pit	...	...	3 "
Tree-hole	...	...	3 "
Calabash	...	...	2 "
Tannery pit	...	...	2 "
Flower pot	...	...	2 "
Dye pot	...	...	1 collection.
Grindstone	...	...	1 "

The full data are given in Table IX.

TABLE IX.

Source.	Larvae.	Jan.	Feb.	Aug.	Sep.	Oct.	Nov.	Dec.	Total.
<i>Barrel.</i>	<i>Culex fatigans</i>	2	...	...	...	1	...	1	4
	<i>Culex nebulosa</i>	3	...	...	1	3	4	6	17
	<i>Aedes argenteus</i>	1	1	...	3	7	4	6	22
	<i>A. argenteus, C. fatigans</i> ...	...	...	...	...	1	...	...	1
<i>Bottle.</i>	<i>Culex nebulosa</i>	2	...	...	...	2	3	...	7
	<i>Aedes argenteus</i>	3	1	...	1	3	4	2	14
<i>Bucket.</i>	<i>Aedes argenteus</i>	6	3	1	3	6	3	1	23
	<i>Culex nebulosa</i>	...	3	...	1	1	...	...	5
<i>Cala-bash.</i>	<i>Culex nebulosa</i>	1	...	...	...	1	...	...	2
<i>Canoe.</i>	<i>Culex nebulosa</i>	...	...	...	...	...	1	...	1
	<i>Anopheles gambiae</i>	...	...	...	...	...	1	...	1
	<i>Culex thalassius</i>	...	...	...	...	...	...	3	3
	<i>Aedes argenteus</i>	...	...	...	...	...	...	1	1
	<i>C. nebulosa, C. thalassius</i> ...	...	...	...	1	...	...	...	1
<i>Catchpil.</i>	<i>Culex fatigans</i>	5	1	...	8	2	1	2	19
	<i>Aedes argenteus</i>	6	1	1	3	4	1	7	23
	<i>Culex nebulosa</i>	40	12	1	5	18	2	11	89
	<i>A. argenteus, C. nebulosa</i> ...	1	...	1	...	...	...	...	2
	<i>C. nebulosa, C. fatigans</i> ...	...	...	...	1	...	...	...	1
<i>Cooler.</i>	<i>Aedes argenteus</i>	1	...	...	2	1	...	...	4
	<i>Culex nebulosa</i>	2	...	...	...	...	...	...	2
	<i>C. nebulosa, A. argenteus</i> ...	...	...	...	...	...	...	...	...
	& <i>C. fatigans</i>	...	...	...	...	1	...	...	1
<i>Drain.</i>	<i>Culex nebulosa</i>	9	3	1	1	1	1	3	19
	<i>Culex fatigans</i>	1	...	...	1	...	...	...	2
	<i>Aedes argenteus</i>	1	...	...	1	...	...	...	2
	<i>Anopheles gambiae</i>	...	...	...	1	1	...	...	1
	<i>Culex decens</i>	...	...	...	...	...	...	1	1
<i>Drum.</i>	<i>Aedes argenteus</i>	7	1	1	4	5	3	10	31
	<i>Culex nebulosa</i>	7	2	1	5	3	...	4	22
	<i>Culex decens</i>	...	1	...	...	...	...	...	1
	<i>Culex duttoni</i>	...	...	...	...	1	...	...	1
	<i>Culex fatigans</i>	...	...	...	...	1	...	...	1
	<i>A. argenteus, C. nebulosa</i> ...	...	...	...	...	...	...	1	1
<i>Dye-pot.</i>	<i>Aedes argenteus</i>	...	...	...	1	...	...	...	1
<i>Flower pot.</i>	<i>Aedes argenteus</i>	...	...	...	1	...	...	1	2
<i>Grind-stone.</i>	<i>Aedes argenteus</i>	...	...	...	...	1	...	...	1
<i>Jug.</i>	<i>Culex nebulosa</i>	4	1	...	...	1	2	...	8
	<i>Aedes argenteus</i>	2	...	...	...	1	1	...	4
	<i>Culex fatigans</i>	...	1	...	...	...	...	...	1
<i>Kettle.</i>	<i>Aedes argenteus</i>	4	2	...	...	...	...	1	7
	<i>Culex nebulosa</i>	2	...	...	...	...	1	...	3
<i>Mortar.</i>	<i>Aedes argenteus</i>	1	...	...	...	1	...	...	2
	<i>Culex nebulosa</i>	...	1	...	...	...	...	1	2
	<i>C. nebulosa, A. argenteus</i> ...	...	...	...	...	1	...	...	1
<i>Pail.</i>	<i>Aedes argenteus</i>	...	...	...	...	2	1	...	3
	<i>Culex fatigans</i>	...	...	...	...	...	...	1	1
	<i>Culex nebulosa</i>	...	...	...	...	...	...	2	2
<i>Pan.</i>	<i>Culex decens</i>	1	...	...	...	...	...	...	1
	<i>Aedes argenteus</i>	...	...	1	2	1	3	1	8
<i>Pit.</i>	<i>Culex nebulosa</i>	...	...	...	...	...	1	...	1
	<i>Aedes argenteus</i>	...	...	...	...	...	1	...	1
	<i>Culex fatigans</i>	...	...	...	...	...	...	1	1

TABLE IX.—*continued.*

Source.	Larvae.	Jan.	Feb.	Aug.	Sept.	Oct.	Nov.	Dec.	Total.
<i>Pool.</i>	<i>Culex fatigans</i> ...	...	...	...	3	2	...	...	5
	<i>Anopheles gambiae</i> ...	...	...	...	3	...	1	1	5
	<i>Aedes argenteus</i> ...	...	...	...	1	2	1	...	4
	<i>Culex nebulosa</i> ...	...	...	...	...	1	1	...	2
	<i>A. gambiae, C. fatigans</i> ...	...	...	...	...	1	...	...	1
<i>Pol.</i>	<i>Aedes argenteus</i> ...	34	23	5	70	122	71	111	436
	<i>Culex nebulosa</i> ...	139	33	4	46	52	49	65	388
	.. <i>fatigans</i> ...	2	...	...	5	...	...	...	7
	.. <i>decens</i> ...	1	...	...	...	...	...	...	1
	.. <i>duttoni</i> ...	...	...	...	...	...	...	1	1
	<i>Anopheles gambiae</i> ...	...	...	...	...	1	2	...	3
	<i>A. argenteus, A. luteocephalus</i> ...	...	...	1	1	...	...	...	2
	<i>A. argenteus, C. nebulosa</i> ...	...	...	1	2	4	...	2	9
	<i>A. argenteus, C. fatigans</i> ...	...	...	...	2	1	1	...	4
	<i>C. duttoni, C. decens</i> ...	...	...	...	1	...	...	...	1
	<i>C. fatigans, C. nebulosa</i> ...	...	...	...	...	...	1	1	2
	<i>C. .. An. gambiae</i> ...	...	...	1	...	...	...	...	1
<i>Tannery pit.</i>	<i>C. fatigans</i> ...	...	...	...	...	1	...	...	1
	<i>Aedes argenteus</i> ...	...	...	...	...	1	...	...	1
<i>Tank.</i>	<i>Culex thalassius</i> ...	1	...	...	...	...	...	...	1
	<i>Anopheles gambiae</i> ...	...	...	...	1	...	...	...	1
	<i>Culex nebulosa</i> ...	...	...	...	...	...	1	1	2
	<i>Aedes argenteus</i> ...	...	...	...	...	...	2	2	4
<i>Tin.</i>	<i>Aedes argenteus</i> ...	17	5	4	4	12	8	6	56
	<i>Culex nebulosa</i> ...	17	3	3	1	7	4	4	39
	<i>Culex fatigans</i> ...	1	...	...	...	1	...	...	2
	<i>Anopheles gambiae</i> ...	...	...	...	...	1	...	...	1
	<i>A. argenteus, C. nebulosa</i> ...	...	...	1	...	...	...	...	1
	<i>A. argenteus, A. luteocephalus</i> ...	...	...	...	...	...	1	...	1
	<i>C. fatigans, C. decens</i> ...	1	...	...	...	...	...	...	1
	<i>C. fatigans, A. argenteus and Eretmopodites chrysogaster</i> ...	..	...	...	...	1	...	...	1
	<i>Aedes argenteus</i> ...	...	...	2	1	2	...	...	5
<i>Tyre.</i>	<i>Culex nebulosa</i> ...	1	...	...	...	...	...	1	2
	<i>A. argenteus, C. nebulosa</i> ...	...	...	1	...	...	...	...	1
	<i>C. nebulosa, C. fatigans</i> ...	...	...	...	...	1	...	...	1
	<i>Aedes luteocephalus</i> ...	...	...	...	1	...	...	...	1
<i>Tree-hole.</i>	<i>Culex nebulosa</i> ...	...	...	...	...	1	...	...	1
	<i>Aedes argenteus</i> ...	...	...	...	...	...	...	1	1
	<i>Aedes argenteus</i> ...	4	...	2	1	2	2	1	12
<i>Well.</i>	<i>Culex decens</i> ...	1	...	...	...	...	..	1	2
	<i>Culex nebulosa</i> ...	2	1	...	...	1	1	...	5
	Totals ...	333	99	33	188	288	184	264	1,389

In the absence of data for the months between February and August it would be unwise to institute comparisons, but the large number of collections received in January and December, these being almost rainless months, would seem to indicate that, in the absence of other breeding-places the mosquito sought the ubiquitous water pot of the usual native household. This utensil harboured every species identified with the exception of *Eretmopodites chrysogaster* which is to be regarded as a "bush" mosquito. As a palliative or accessory measure the indications are that sanitary efforts concentrated on the water-pot, which the introduction of a pipe-borne supply does not seem to have reduced materially in numbers, would be very usefully directed, during the dry season, in reducing the number of household mosquitos.

BLOOD-SUCKING INSECTS SENT FOR IDENTIFICATION.

From Kano—

Cimex lectularius and *C. rotundatus*, about 150 of each.

From Onitsha—

Taeniorhynchus (*Mansonioides*) *africanus*, 86 ♀ ♀.
Culex invidiosus, 1 ♀.

From Mamfe—

<i>Chrysops silacea</i>	...	...	...	...	18 ♀ ♀.
<i>Glossina palpalis</i>	...	...	...	...	26 ♀ ♀.
,, <i>pallicera</i>	...	...	...	...	4 ♀ ♀.
,, <i>tabaniformis</i>	...	...	...	...	1 ♀.
<i>Tabanus kingsleyi</i>	...	...	...	...	8 ♀.
<i>Simulium damnosum</i>	...	...	...	...	182 ♀ ♀.
<i>Culicoides</i> sp.	...	...	...	...	numerous.

(All the above insects were dissected, and larval filariae similar to those described by Blacklock, were found in five specimens of *Simulium damnosum*, the parasites being present in the thorax only, in two, in both head and thorax in two and in the proboscis only in one).

From Makurdi—

<i>Anopheles gambiæ</i>	...	...	...	12 ♂ ♂,	12 ♀ ♀.
<i>Culex univittatus</i>	...	...	...	4 ♂ ♂,	7 ♀ ♀.
<i>Lutzia tigripes</i>	...	...	...	4 ♀ ♀.	
<i>Anopheles squamosus</i>	...	...	...	2 ♂ ♂,	1 ♀.
,, <i>maculipalpis</i>	...	...	...	3 ♂ ♂,	3 ♀ ♀.
<i>Aedes argenteus</i>	...	...	...	1 ♀.	

From Lagos—

<i>Aedes irritans</i>	...	...	...	3 ♂,	13 ♀ ♀.
,, <i>nigricephalus</i>	...	...	...	2 ♀ ♀.	
,, <i>argenteus</i>	...	...	...	3 ♀ ♀.	
<i>Culex thalassius</i>	...	...	...	5 ♀ ♀.	
,, <i>insignis</i>	...	...	...	3 ♀ ♀.	
,, <i>fatigans</i>	...	...	...	1 ♀.	
,, (<i>culiciomyia</i>) <i>nebulosa</i>	...	...	...	3 ♀ ♀.	
<i>Glossina palpalis</i>	...	...	...	1 ♀.	
<i>Tabanus secedens</i>	...	...	...	2 ♀ ♀.	

Note on the ova of Aedes argenteus—

Some eggs, dried on filter paper, of *Aedes argenteus* were taken to England on 12th February, 1926, and were brought back again to Lagos on 18th August, 1926. They were placed in water on 14th September, 1926, and on 19th September, 1926 many larvae hatched out and three adults emerged.

A note on cross-breeding experiments—

Aedes argenteus, female, and *Culex thalassius*, male, negative.

Aedes argenteus, female, and *Aedes luteocephalus*, male, eggs laid but did not hatch.

Aedes argenteus, female, and *Culex fatigans*, male, eggs laid but did not hatch.

DISSECTIONS.

Taeniorhynchus africanus,
sixty dissected one showed numerous amoebae in
stomach contents, others negative.

Aedes domesticus, fourteen
dissected two showed peculiar cysts in
stomach contents, others negative.

Anopheles umbrosus, four
dissected two with filaria larvae in thorax,
others negative.

Culex duttoni, three dis-
sected all negative.

Anopheles gambiae, three
dissected one with filaria larvae in thorax,
other two negative.

Culex nebulosa, two dis-
sected both negative.

Aedes nigricephalus, two dis-
sected both negative.

Lutzia tigripes and
Culex thalassius
one of each dissected ... both negative.

